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THE DIAGNOSIS AND TREATMENT OF UPPER RESPIRATORY DISEASES

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In order to understand clearly some of the factors involved in upper respiratory infections, it is necessary to begin with the early development of the nose. In the fetus, even at one hundred and twenty days, we begin to see definite nasal formation and distinct cavities of some of the nasal accessory sinuses. At birth the antrum occupies a small oval space to the inner side of the orbit. Its later development is downward, and it assumes its full shape after eruption of the permanent teeth. The maximum development is attained between the fifteenth and eighteenth years. The ethmoidal sinuses are present at birth, but assume little importance until the second or third year of life. The frontal and sphenoid sinuses make their appearance in the third year. So we find that in earliest childhood the nose and its related sinuses are developed sufficiently to become foci of infection.

The nasal mucosa, continuing into the related accessory sinuses, is particularly constructed to combat the invasion of offending organisms. The ciliated epithelium of the mucous membrane has a definite wave-like action to repel invading micro-organisms. The submucosa contains goblet-like glands which produce an antiseptic, mucoid substance to incorporate germs and irritating foreign particles into a gelatinous mass which is usually diverted to the intestinal tract.

It is seen, then, that the first line of defense against infection of the upper respiratory tract is the anatomical construction of the nose and its physiological reactions. If these are normal, their functions can be effectively carried out. In this region there is found the greatest formation of lymphatic glands to prevent invasion of organisms into deeper structures. A well-developed chain of resistance here often prevents serious conditions in other parts of the body. However, as in any other organ, if pathological changes occur, local resistance to invading organisms is lessened.

Our first duty begins with the infant and the growing child. Diet must be given the greatest consideration. Every child should be given the best possible opportunity to begin life with a strong, healthy body which is able to overcome early infections by natural resistance.

The Common Cold

In any discussion of acute upper respiratory infections due consideration must first be given to the so-called "common cold". For many years scientists have been working to determine the exact etiological factors of the common cold. At the present time this problem still remains unsolved, although much valuable information has been accumulated in recent years concerning the bacterial nature of this disease.

The condition generally referred to as cold is characterized clinically by hyperemia, congestion and disturbances in secretion of the mucous membranes lining the nose, throat, and bronchi. Symptoms of dryness, irritation, cough and obstructed breathing are usually present, while general symptoms

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of aching, malaise, headache and fever are frequently associated with them.

For the purpose of description the classification given by Cecil[1] is used: non-contagious colds, contagious colds, and influenza.

Non-contagious colds are produced by some local reaction in the mucous membrane, whereby its normal power of resistance is so lowered that endogenous organisms may enter the tissues. Vasomotor changes, systemic disturbances, local foci of infection, heat, cold, changes of clothing, injuries and irritation of the mucosa by chemical agents such as chlorine and bromine all play an important part in this type of cold.

In contagious colds, the infecting agent is exogenous and is usually some form of virulent organism, such as the pneumococcus, the streptococcus or the influenza bacillus. Some contend that the initial cause is a filtrable virus, and that complications are due to the invasion of secondary organisms.

The influenzal form may be endemic, epidemic or pandemic.

There is little exact information at this time regarding the true etiology of this disease. Many maintain that a filtrable virus is the cause, while others consider Pfeiffer's bacillus the important organism. Studies of the bacteriology of the upper respiratory tract have engaged the attention of many investigators. It has been found[2] that the micro-organisms appear on the mucous membrane of infants about twelve hours after birth, and from this time on are always present. The bacterial flora at this early period is comparatively simple, being composed of organisms which are introduced while nursing. Those usually found are Staphylococcus albus, Staph. aureus, Micrococcus tetragenus, a few non-hemolytic streptococci and transients of variable characteristics. As time goes on many other organisms appear in this region. Quite a number are pathogenic; others may become so under certain conditions. A certain group adapt themselves to growth on the mucosa and become intimately associated with it.

Bloomfield[3] is of the opinon "that cultures made repeatedly from the throat of a healthy individual over a considerable length of time reveal two groups of organisms. In the first place there is a group of non-hemolytic streptococci and gram-negative cocci, which is constantly present and seems to constitute the true normal flora of the throat in the sense of actually living its complete life history in that environment. In addition to this group many other organisms may be recovered from normal throats, but their presence seems to be only transient. They disappear in a few days, as a rule, just as foreign organisms do when experimentally introduced."

In a bacteriologic study of 63 cultures of the chief flora in the rhinopharynx, Myer Solis-Cohen[4] found, in order of frequency: Micrococcus catarrhalis (57 per cent), Staphylococcus pyogenes albus (54 per cent), pseudo-diphtheria bacillus (30 per cent), Streptococcus viridans and Staphylococcus pyogenes aureus (27 per cent), non-hemolytic streptococcus (24 per cent), pneumococcus (21 per cent), hemolytic streptococcus (19 per cent), unclassified staphylococci (11 per cent), and Friedlander's bacillus (8 per cent). To this list could be added many other organisms which have been recovered from this region at different times by other observers. With such a variety of organisms capable of producing variable reactions it is not surprising that infection is so common in the upper respiratory tract. However, there are certain factors which control the action and elimination of bacteria in this region.

One of the most important is the anatomical construction of the nose and throat. The nose is particularly developed to prevent the entrance of organisms into the deeper air passages. Hasslauer[5], and Thompson and Hewlet[6] have demonstrated that many infected bacteria are arrested by the moist vibrissae at the anterior nares, and are kept away from the inner recesses of the nose. When air is inspired the current strikes against the turbinate bones, where it is broken up and deflected in all directions. The moist film of mucus covering the mucosa is especially prepared to incorporate remaining organisms so that they can be carried backward to the throat by the action of ciliated epithelium.

1. Cecil, R. L.: Common Cold, Its Prevention and Treatment, M. Clin. N. America 8:103 (July 19) 1924.
2. Bloomfield, Arthur L.: Adaptation of Bacteria to Growth on the Human Mucous Membranes, With Special Reference to Throat Flora of Infants, Bull. Johns Hopkins Hosp. 30:61 (Feb.) 1922.
3. Bloomfield, Arthur L.: Significance of the Bacteria Found in the Throats of Healthy People, Bull. Johns Hopkins Hosp. 32:33 (Feb.) 1921.
4. Solis-Cohen, M.: Rhinopharynx as a Site of Focal Infection, Ann. Otol, Rhinol, and Laryngol. 33:935 (Sept.) 1924.
5. Hasslauer: Centertalle-Bakterioe, I Obt. Ref. Bd. 37:1, 1906.
6. Thompson and Hewlet, in Lancet, 1:86, 1896.

In the mouth many bacteria are eliminated by the flushing action of the secretions. This is assisted by muscular contractions in the throat during the act of swallowing, so that organisms are rapidly directed backward into the esophagus. The value of tenacious mucus in holding organisms is also an important factor here. The bactericidal action of saliva[7] on certain organisms is of some importance.

The normal resistance of the epithelial layer of mucous membrane, combined with the phagocytic action of the underlying vascular supply, is of considerable importance in preventing the invasion of organisms. However, when there is some disturbance in the protective mechanism, such as is often produced by vasomotor changes, local irritation, and injuries of the mucosa (which may be of mechanical, chemical or thermal origin), the susceptibility to bacterial invasion is much greater. The incidence of infection is also influenced by the number, character and virulence of the offending organism. Many theories about the relation of various bacterial factors in the etiology of the cold have been offered. McCallum[8] feels that chilling of the skin predisposes in some way to infection by the usual organisms found on the mucous membrane during health. Osborne[9] considers colds contagious and believes that many different germs may be responsible. Mudd, Grant and Goldman[10] are also inclined to this view.

Bloomfield[11] found that there is a marked similarity in the throat flora of normal persons and those suffering with colds. He does not feel that the usual throat inhabitants, such as staphylococci, streptococci, pneumococci or the influenza bacilli, cause colds, but that their development is made more favorable during an attack, and that they produce secondary complications. Jordan, Norton and Sharp[12] found the throat flora in patients with colds to be similar to that found in health. Williams, Nevin and Gurley[13] made a like observation. They also reported that 39 per cent of the patients with colds had pneumococci as predominating organisms, as compared with 26 per cent of healthy subjects. Gordon[14] found pneumococci in 35 per cent of patients with colds and in 21 per cent of healthy persons examined.

In 1914, Kruse[15] diluted nasal secretions of a patient with a common cold and introduced them into the noses of thirty-six volunteers. He claimed that 42 per cent became ill with the disease. Kruse believed that the exciting agent was a filtrable micro-organism, although he was unable to demonstrate the germ. Foster[16] attempted to confirm the experiments of Kruse at a later date. He not only agreed with Kruse, but also claimed to have cultivated a minute anaerobic micro-organism similar to the "globoid bodies" of Flexner and Noguchi[17]. Experiments on the transmission of colds from man to man were carried out by Olitsky and McCartney[18]. They filtered the nasal secretions of infected patients and produced symptoms of cold in four men out of six in the test.

From this conflicting maze of opinions it is obvious that the specific agent causing colds has not been agreed upon. However, the mass of evidence indicates that sooner or later all colds can be considered infections[19]. This, after all, is the most important point for us as practical clinicians. With this knowledge we can advise the necessary precautions in preventing the spread of this most common disease and can outline specific measures for prophylaxis and treatment.

The public should be more carefully instructed as to the contagiousness of this infection. People should be made to realize that sneezing, coughing or spitting in public places is a pernicious practice. A simple globule of mucus from a carrier may contain many organisms which are pathogenic to some susceptible person, while producing no

7. Bloomfield, Arthur L.: Mechanism of Elimination of Bacteria From the Respiratory Tract, Am. J. M. Sc. 164: 854 (Dec.) 1922.
8. McCallum: Textbook of Pathology, Philadelphia, W. B. Saunders, 1918, p. 370.
9. Osborne, in New York State J. Med. 109:529, 1919.
10. Mudd, S.; Grant, S. B.; and Goldman, A.: Etiology of Acute Inflammations of the Nose, Pharynx, and Tonsils, Ann. Otol., Rhinol., and Laryngol. 30:1 (March) 1921.
11. Bloomfield, Arthur L.: Variations in Bacterial Flora of Upper Air Passages During the Course of Common Colds, Bull. Johns Hopkins Hosp. 32:121 (April) 1921.
12. Jordan, E. O.; Norton, J. F.; and Sharp, W. B.: The Common Cold; Influenza Studies, J. Infect. Dis. 33:416 (Nov.) 1923.
13. Nevin, M.; Gurley, C. R., and Williams, A. W.: Methods of Demonstrating Micro-Organisms, J. Immunol. 6:5 (Jan.) 1921.
14. Gordon, J. E.: Relationship of Pneumococcus to Acute Infections of the Upper Respiratory Tract in Man, J. Infec. Dis. 29:437 (Nov.) 1921.
15. Kruse, W., in Muenchen. med. Wchnschr, p. 1547, 1914.
16. Foster, G. B., Jr.: The Etiology of Common Colds, J. A. M. A. 66:1180 (April 15) 1916; J. Infec. Dis. 21:451, 1917.
17. Flexner, S., and Noguchi, H., in J. Exper. Med. 18, 1913.
18. Olitsky, Peter K., and McCartney, James E.: Studies on Naso-Pharyngeal Secretions From Patients With Common Colds, J. Exper. Med. 38:427 (Oct.) 1923.
19. Looper, Edward A.: The Bacteriologic Factor in the Cause of Common Colds, South. M. J. 18:143 (Feb.) 1925.

symptoms in the host. The average person is indifferent to the ordinary cold and does not realize how many complications follow in its wake. Many cases of otitis media, mastoid infection, sinus disease, bronchitis and pneumonia could be prevented, if treatment was enforced during an attack.

The general death rate each year is much higher during January, February, and March, when colds are more prevalent. Improper ventilation, exposure to bad weather, and closer association of people during this time contribute to the spread of this infection.

Another point which emphasizes the bacterial nature of the common cold is the development of immunity following attacks. It is true the immunity may be of short duration, usually lasting about six to eight weeks. In the interim the patient can take unusual chances without developing symptoms. This short period of immunity can be explained as due to the localization of infection, which is limited to the mucous membrane, and to the low virulence of the organism causing the disease.

With the knowledge that immunity can be produced, many efforts have been made successfully to use vaccines in the treatment of colds. Autogenous vaccines of mixed cultures have given the best results, although stock vaccines have been used with some apparent success. The whole field of vaccine therapy is an interesting one for development. Since the use of chemotherapy the sulfonamides have been used with much success in the treatment of upper respiratory infections. Internal administration, supplemented by local application of the drug, has been very satisfactory in the majority of patients.

The Nasal Accessory Sinuses

It is generally agreed that symptoms of the common cold continuing for a period of more than two weeks mean that there is involvement of the nasal accessory sinuses. This is particularly true in infants and young children. Children with infected sinuses are irritable, anemic, and undernourished, and present all the symptoms associated with an active focus of infection. Furthermore, such infections retard the development of the sinuses themselves, creating an ideal condition for the continuance of a chronic disease.

It is a common practice to attribute all chronic colds and all cases with nasal discharge, regardless of the location, character or amount of secretion, to diseases of the tonsils and adenoids. It is often taken for granted that this is the only factor to be considered, although the true source of infection may be in the paranasal sinuses. We frequently encounter children who have had their tonsils and adenoids removed, but who do not get the desired relief from their symptoms. They state that their colds are as frequent and the nasal discharge as constant and as severe as before the operation. A situation of this kind can be avoided if more attention is paid to the sinuses before the tonsils and adenoids are removed. In all children with inflammatory lesions around the nose and throat, with the nasal symptoms predominant, the sinuses should be examined. Where there is a great deal of sneezing, congestion of the turbinates, and constant colds with thick, profuse nasal discharge, a careful investigation of the sinuses is called for. If the diagnosis is correctly made, treatment can be properly instituted and the results will be satisfactory.

While children are not as susceptible to diseases of the sinuses as are adults, the condition is frequent enough to require careful consideration. In fact, the subject requires more attention than is generally given to it by rhinologists. Haike[20] made an autopsy study of 394 children. He opened the sinuses of 62 subjects and found them diseased in 52 cases. The ages varied from 9 months to 13 years. Forty-seven cases had an infection of the maxillary antrum, 2 of the sphenoids, and 3 of the ethmoid cells. Dean and Armstrong[21] investigated the sinuses in a group of children presenting the common symptoms of infected tonsils and adenoids. One hundred and forty-five cases were examined, and of this number 65 showed definite involvement of the sinuses. In a similar group of cases White[22] made x-ray studies of 50 children who had been admitted to the hospital for tonsillectomy

20. Haike: Beitr. z. pathol. Therap. d. oberen Atmungswege, Wiesbaden.

21. Dean, L. W., and Armstrong, M.: (a) Some Indications for Operation on the Nasal Accessory Sinuses in Children, Laryngoscope 31:273 (May) 1921; (b) Diseases of the Accessory Sinuses in Infants and Young Children, Including Bacteriologic Study, Ann. Otol., Rhinol. and Laryngol. 28:452 (June) 1919; (c) Occasional Necessity of Doing Radical Operations on Paranasal Sinuses in Children, With Report of Cases, Ann. Otol., Rhinol. and Laryngol. 29:602 (September) 1920.

22. White, F. W.: Pathologic Nasal Accessory Sinuses in Children, Ann. Otol., Rhinol. and Laryngol. 30:221 (March) 1921.

and adenoidectomy, and out of the 50 cases 41 showed pathologic sinuses.

In the early years of childhood the upper respiratory tract is particularly susceptible to infection and is generally involved in most of the exanthematous diseases. In practically every case of measles, whooping cough, diphtheria and scarlet fever, severe reactions are produced in this area. We are always on the alert for ear involvement following these diseases, but do we pay sufficient attention to nasal complications?

The sinuses are lined with mucous membrane similar in structure to that of the eustachian tube, and by the process of continuity can be readily infected. The numerous recesses and cavities adjoining the nasal fossae provide an ideal location for the implantation and development of organisms. If bacteria are placed upon the smooth, unobstructed mucous membrane of the mouth we know that they will be eliminated by the normal douching action of the oral secretions, as demonstrated by Bloomfield's work[23]. However, when they lodge in obstructed crevices such as the tonsillar crypts, reduplicated nasal mucous membrane, or sinus cavities, they remain for a much longer time. If the body resistance is below normal, or if the predominating organisms are of a very virulent strain, the tissues may not be able to resist infection. When the sinuses become extensively infected, eradication of the disease is extremely difficult. Congested nasal mucosa, hypertrophied turbinates, septal deviations, and nasal cavities interfere with ventilation and drainage, thus prolonging a condition which could be easily eradicated in a more accessible environment.

Diagnosis

By early diagnosis and the application of conservative treatment, many extensive operative procedures can be avoided and serious complications can be prevented.

A careful history is necessary in making an early diagnosis. Repeated nasal infections or so-called common colds lasting more than two weeks usually indicate some involvement of the sinuses. Discharge may or may not be present, depending upon the amount of drainage. In children the diagnosis is much more difficult, because subjective symptoms can not be depended upon. Careful inquiry should be made to determine if a child has had measles, scarlet fever, diphtheria or whooping cough, as the exanthematous diseases are usually complicated by severe nasal infections and sinus involvement.

In adults, the acute cases usually cause definite symptoms, such as pain, tenderness, headache, nasal discharge, and difficulty in breathing, which make the diagnosis comparatively easy. In chronic infections quite a different problem is presented. A certain number of cases show such definite local symptoms that a diagnosis is easily made, and this group gives us little trouble. It is the borderline case, with few, if any, helpful localizing symptoms, which gives us most concern. The cases in which the nasal symptoms are insignificant or are masked by certain general symptoms of toxic absorption, which divert our attention along other lines, are perplexing. This is especially true of cases with arthritis, indefinite rheumatic pains, digestive disturbances, malaise, headache, and vertigo.

Considering the anatomical structure and close relationship of the sinuses to other important structures, it is not strange that the most prominent symptoms come from these special organs. The most typical examples are eye lesions, cranial involvement, and reflex nervous phenomena.

During the past few years I have seen about 25 patients with distinctive manifestations of cerebral irritation caused by cerebral edema resulting from the absorption of toxins from a low-grade and unsuspected sinus infection. Some of these patients entered the hospital in a state of coma, and several had convulsions. All of the symptoms quickly cleared up with simple drainage, and the patients have remained cured.

In the diagnosis of these obscure cases, every available aid is required, and good x-ray plates are most important. The use of lipiodol makes them still more helpful. While entire dependence should not be placed on an x-ray plate, it is our most reliable objective method of examination. A clear plate will reveal the shape and size of the sinuses (whether they are fully developed, infantile, or walled off by septa) and the pathological state (hyperemia, a thickened membrane or empyema). It will also show foreign bodies such as teeth, as well as single or multiple polypi.

Every rhinologist should read and study his own x-ray plates. In my office I have my

23. Looper, Edward A.: *Infections of the Nasal Accessory Sinuses in Children*, M. Clin. N. America 21:1323 (Sept.) 1937.

own x-ray equipment, and find it very convenient and satisfactory. Lipiodol injections can be made easily. Plates taken and developed by a technician can be presented to me in a short time for interpretation and study. In every patient who gives a history of recurrent nasal infections I make an x-ray of the sinuses, and it is surprising how many cases are discovered which, if clinical symptoms alone were depended upon, would have been overlooked[24].

Transillumination is unsatisfactory at best, and can not be depended upon for accurate diagnosis. Tenderness over the sinuses is not a constant symptom and is generally absent in the low-grade infections. Bacteriological smears and cultures do not give the desired information, because the upper respiratory tract is the natural habitation of many organisms, and it is difficult to determine when an area is actually infected.

We place most dependence upon the examination of the intranasal structures for abnormal changes; but, unfortunately, in many of these cases it is impossible to determine the presence or absence of infection by the local appearance of the tissue. The nasal mucosa may seem to be normal, the breathing space unobstructed, the turbinates of regular size, with little change in nasal secretion, while the patient has definite general symptoms of toxic absorption.

Treatment

A knowledge of the anatomical shape and position of the sinuses and the location of the ostia is important in treating sinus infections. The frontal sinuses being above, with their ostia at the most dependent portion, it is easy for this sinus to drain. This fact explains its satisfactory response to local applications and suction treatment, if treatment is instituted early.

Because of their location and large size, and the fact that their ostia are located at the upper inner boundary, spontaneous drainage of the antra, when once they are filled with pus is difficult. At best, no drainage can occur until the antral cavity overflows. Local astringent applications are of little use. The logical method of treatment is to establish drainage at the most dependent part, through the inferior meatus, by a simple trocar and to irrigate the antrum with warm saline solution. Unless this is done the condition becomes chronic, and the antral cavity acts as a reservoir for pus. In time the membrane becomes diseased, and more radical measures are necessary to effect a cure. The sphenoids and ethmoids are more amenable to local treatment than are the antra.

The ethmoidal labyrinth is very susceptible to infection, and, on account of its honeycomb-like arrangement of cells, the infection is very difficult to eradicate after it has once obtained a foothold. The turbinates, because they are susceptible to hypertrophy and deviations of the septum are common, play an important part in the etiology and treatment of sinus diseases in this area. Surgical measures are often necessary to obtain ventilation and free drainage. Treatment, naturally, must be governed by the type of infection.

Most of the acute sinus infections respond quickly to treatment, and, as a rule, the prognosis is good. The local treatment consists in cleansing the nose with some warm alkaline solution such as normal salt or Dobell's solution, which frees the mucous membrane of secretions and a certain number of bacteria. After this, some astringent solution such as adrenalin or ephedrine and cocaine is applied. This shrinks the congested tissue and allows a certain amount of drainage. It is followed by a bland, soothing, oily spray. In acute cases we are often able to help the condition with suction treatment. The use of solutions of chloretone inhalant or ephedrine inhalant in croup kettles is soothing as well as beneficial.

Another type of sinus infection which is very common and is often associated with acute sinus conditions is known as vacuum frontal sinusitis, and is produced by congestion and hypertrophy in the upper nasal passages, so that the nasofrontal duct is closed. The air is absorbed in the sinus and a certain amount of suction is produced, with resulting hyperemia of the mucous membrane. This causes excruciating pain and headache. The treatment consists of shrinking the congested mucous membrane or removing the obstruction by operation. This condition is similar to an acute catarrhal otitis media, and the principle of treatment is similar.

Chronic diseases of the accessory sinuses are often neglected. Patients are often

24. Looper, Edward A.: Infection of the Nasal Accessory Sinuses, West Virginia M. J. 25:710 (Dec.) 1929.

given the impression that they have a "chronic catarrh" which is incurable. This idea is remarkably prevalent among the laity, and many unfortunate victims are suffering who might be helped by proper treatment. Quite a number of these cases can be cured by local treatment, no operative procedures being necessary. In such cases we find suction very efficacious. Many times, however, no results can be obtained by local treatment, because the mucous membrane of the sinuses is so badly diseased that nothing short of extensive operations will eradicate the condition. This is often true in antral disease. Fortunately, most of the obstinate cases can be cured by a radical operation, the type of operation being determined by the etiology and duration of the disease.

Most cases of frontal sinus infection can be cured by conservative intranasal surgery. The nasofrontal duct being at the inferior wall of the sinus, drainage is not difficult after obstructions such as spurs, septal deviations or hypertrophied turbinates have been removed.

There are a certain number of cases which resist all efforts to effect a cure by intranasal surgery. With this group some external procedure is required. In the purely chronic cases, there is usually sufficient time to choose and perform any one of the classical operations which the operator likes best. In a small group, however, the development of symptoms and complications is so rapid that the quickest and safest form of drainage is required at once.

Complications

Orbital abscess is one of the most frequent and often the most dangerous complication of accessory sinus disease. A definite abscess is characterized by edema of the affected eye, lids and surrounding tissue. The eyeball is forced out and there is immobility of the extra-ocular muscles. With pus formation, there is elevation of temperature and increased leukocytosis. If the pressure is not relieved by free drainage vision may be affected. Many of these cases occur in early life.

Meningitis and brain abscess are serious complications of sinus disease and frequently terminate fatally. Fortunately, such cases are rare. The complications associated with any chronic focus of infection may be present with accessory sinus diseases in children. Anemia, loss of weight, low, unexplained temperature, and gastrointestinal symptoms are often present. Any such group of symptoms calls for investigation and treatment of the sinuses.

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THE TREATMENT OF RESPIRATORY DISEASES IN CHILDREN

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Respiratory diseases are responsible for over one-third of the illness seen in private practice[1] and in hospitals[2], and are especially important during the present war. They caused nearly twice as many deaths among American soldiers and sailors during World War I as did battle casualties[3] (table 1). Unfortunately preventive measures, except in tuberculosis, diphtheria and pertussis, have had little effect.

Table 1. Comparison of Deaths from Respiratory Conditions and Battle Injuries in the U. S. Army and Navy During World War I[3].

Cause	*No. Deaths*
Measles*	2,482
Rubella*	82
Diphtheria	225
Influenza	28,822
(Total Respiratory)	(31,611)
Battle Injuries	16,961

* Deaths due to respiratory complications.

The signs of respiratory disease—sore throat, stridor, cough, nasal discharge, epistaxis, dyspnea, rales and dullness—can be caused by thirty common and twenty rare conditions. Obviously, a physician cannot take into consideration all fifty every time he sees a patient with a "cold", but he should consider the twenty-seven in table 2, and concentrate on the more likely possibilities. He especially should consider the conditions for which adequate therapy is available, and not, as is the increasingly frequent practice, postpone the diagnosis until he has tried sulfonamide therapy for a few days; even though the sulfonamides are helpful in all respiratory infections except tuberculosis and diphtheria.

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1. London, A. H., Jr.: The Composition of an Average Pediatric Practice, J. Pediat. 10:762-771 (June) 1937.
2. Arena, J. M. and Harriss, R. R.: The Frequency and Distribution of Diseases in Children, South. Med. and Surg. 97:520-522 (Sept.) 1935.
3. Davison, W. C.: The Reduction of Communicable Diseases Among Troops and Children During National Defense Program, War Med. 1:863-872 (Nov.) 1941.

Table 2. Respiratory Diseases in Children

Upper respiratory tract	*Lower respiratory tract*
Diphtheria	Bronchitis
Rhinopharyngitis	Pneumonia
Tonsillitis	Empyema
Laryngitis	Tuberculosis
Tracheitis	Pleurisy
Influenza	Foreign body
Otitis media	Lung abscess
Mastoiditis	Bronchiectasis
Sinusitis	Atelectasis
Cervical adenitis	Pneumothorax
Peritonsillar abscess	Psittacosis
Pertussis	Pleurodynia
	Fungus infections

Allergy

Hay Fever
Asthma

Diphtheria is the most important respiratory disease. It should be eliminated by immunization of all infants at the age of 9 months, but it is entirely too common and too frequently mistaken for a "sore throat" by the family and sometimes by the physician. Among 10 diphtheritic patients treated at Duke Hospital during one month, 8 had not been seen by a physician for an average of five days[4], and of 9 other children suffering from diphtheritic paralysis during the preceding year, 7 were not taken to physicians until the onset of paralysis[5]. Every child who has hoarseness, bloody nasal discharge, laryngeal obstruction, dyspnea, or a membrane on the pharynx or larynx should, *without waiting for a report from a throat or nose culture or smear,* have 0.1 cc. of horse serum (diluted 1:10 with saline) injected intradermally, and if no redness appears within thirty minutes, should have an intramuscular injection of 10,000 units of diphtheria antitoxin[6]. This should be repeated twenty-four hours later if the infection continues to spread, or recurs later. If the patient is sensitive to horse serum (as indicated by the appearance of a red wheal within thirty minutes), or has had asthma, eczema or hay fever, he should be desensitized by having small amounts of the diphtheria antitoxin given every thirty minutes. Far less harm is done by the unnecessary administration of diphtheria antitoxin than by delay. If fusiform bacilli and spirochetes are present, neoarsphenamine or sulfarsphenamine should be injected intramuscularly, because the patient may have Vincent's angina, and not diphtheria. Sulfonamide therapy[7] is helpful for associated streptococcal and staphylococcal infections but *should not be substituted* for antitoxin. In severe (malignant) diphtheria, 100 cc. of sterile 25 per cent intravenous dextrose should be given very slowly, in addition to antitoxin, with or without insulin, four to six times daily; daily blood transfusions should also be given. A Schick test should be done three months after recovery, and if it is positive, the child should be immunized.

4. Davison, W. C.: The Medical and Hospital Facilities Available to Children in North Carolina, North Carolina M. J. 2:345-348 (July) 1941.

5. Arena, J. M. and Rasmussen, L. P.: Diphtheritic Polyneuritis, J. Pediat. 13:352-356 (Sept.) 1938.

6. Patients with marked laryngeal symptoms should be sent to a hospital **before** the antitoxin is given, because in these patients a tracheotomy may be necessary within a few minutes after the serum has been injected. To try to make a clinical differentiation between laryngeal diphtheria and "croup" or streptococcus laryngitis may be dangerous for the child; he should be sent to the hospital, given diphtheria antitoxin, placed in a steam tent or near a humidifier, and treated by suction or, if necessary, tracheotomy. The absence of diphtheria bacilli or of a membrane in the throat does not rule out diphtheria; throat and nose cultures may be negative in proven cases of diphtheria.

7. The choice of the drug, the dosage, the effective blood level, and the reactions depend on the patient and the infecting organism. Daily urinalyses (especially for red blood cells) and white and red blood cell counts, and the determination of the blood or urine concentration of the drug every two days, **and** bactericidal tests of the patient's organism with various sulfonamides are necessary. Spectroscopic examination of the blood for sulfhemoglobin and methemoglobin are required only when cyanosis is produced by sulfanilamide. The drug or dosage should be changed in accordance with these tests, or if hematuria or reactions occur, or the patient does not improve. The drug usually should be continued as long as the white blood cell count is elevated and a left Schilling count persists. As agranulocytosis may appear after the drug is stopped, a white blood cell count one month later is advisable. Sodium sulfapyridine and sulfadiazine have the widest range of bactericidal effect against organisms; sulfanilamide usually, but not always, is more effective in meningococcal and streptococcal infections, and diffuses better than the other drugs into the spinal fluid; sulfapyridine generally is better in the pneumococcal, and sulfathiazole in the staphyloccal and dysenteric varieties. Sulfadiazine diffuses into the pleura, peritoneum and spinal canal.

The following dosage usually will maintain an effective blood concentration of 10 mg. of sulfanilamide and sulfadiazine per 100 cc., 8 mg. of sulfapyridine, 5 mg. of sulfathiazole, and 1 or 2 mg. of sulfaguanidine. An initial dose of 1 to 3 Gm. (15 to 45 grains) should be given orally or rectally, followed by 0.2 to 0.5 Gm. (3 to 7.5 grains) every four hours. One year old infants require a **daily total** (divided into four to six doses) of 1 Gm. (15 grains); 2 year old children 2 Gm. (30 grains), and 3 year and older children 3 Gm. (45 grains), in **addition** to an initial dose of these same amounts. If the

"Common cold", coryza, rhinitis, rhinopharyngitis, tonsillitis, laryngitis or croup, tracheitis, and influenza: As may be seen in figure 1, capsules of codeine and papaverine (each 16 mg., or ¼ grain) three times a day after meals for the first two days after onset reduce the length and complications of a "cold"[8]. Sulfonamide therapy[7] will prevent secondary infections[9]. The constipation caused by codeine may require treatment. Bedrest for forty-eight hours, large amounts of fluid, nose and throat sprays of 0.25 per cent aqueous neosynephrin or 1 per cent aqueous racephedrine (the nasal secretion first being removed with a soft rubber ear syringe), Penetro or Benzedrine inhalers (not after 4 p.m.), gargles of 0.6 Gm. (10 grains) of acetylsalicylic acid or 1/5 teaspoonful each of table salt, baking soda and borax in a glass of warm water, and hexylresorcinol and other throat lozenges are helpful. Sedatives also are useful, either alone, or added to cough mixtures, if the cough causes vomiting or loss of sleep. Sedatives, however, should be used *only* if the cough is irritating, painful and nonproductive; both sedatives and cough mixtures are contraindicated if the cough is loose and productive, as lung abscess or bronchiectasis occasionally may be caused by the accumulation of "uncoughed-up" secretion. An ice pack or collar to the throat sometimes relieves a severe sore throat or hacking cough. Cough due to a swollen or enlongated uvula is helped by honey or saturated sugar solution; the osmosis reduces the edema. If the cough is very troublesome, a 1 per cent butyn throat spray and a humidifier or steam tent may be used. Other measures are worse than useless, especially nasal irrigation, oil nose drops which may cause lipoid pneumonia, argyrol nose drops or packs, and throat painting with silver nitrate or other antiseptics. The diet need not be changed. Cathartics have no influence on colds, except that the patient dare not cough if his bowels are loose.

Otitis media: For the first forty-eight hours, unless the child is extremely ill, a hot water bottle over the ear, a nasal spray of 1 per cent aqueous racephedrine, and two to four drops of atropine (or ephedrine) and chlorobutanol in glycerin[10], or of 5 per cent phenol in glycerin should be instilled in each ear every six to eight hours. Not more than six instillations should be given, and they should never be given after the drum is opened. Oral sulfonamide therapy[7] shortens the course of the inflammation and reduces the risk of mastoiditis, although it may mask the signs. The drum should *not* be incised unless it is gray and bulging with obliterated landmarks and the patient is very ill, restless and cannot sleep. If the drum is opened, the canal should be irrigated every four to eight hours for the next two to six days with 3 per cent sulfadiazine in 8 per cent triethanolamine, or with one or two ear syringefuls of ear lotion[11], and swabbed dry. *Chronic otitis* should be treated by blowing an ear-syringe of 1 per cent iodine-boric powder through the perforated drum

above blood concentrations of the drug are not produced—and often the size of the dose and the blood level are not correlated—this dosage should be increased or decreased to produce these concentrations; higher levels are necessary in severe infections. If the infection is very severe, or oral or rectal administration is impractical, sodium sulfapyridine, sodium sulfathiazole or sodium sulfadiazine may be given intravenously or subcutaneously in **daily total amounts** of 6 cc. of a 0.5 per cent solution in sterile normal saline (5 mg. per cubic centimeter) per kilogram of body weight (2.5 cc. per pound). These solutions cannot be autoclaved. Similar amounts of 0.8 per cent sterile sulfanilamide (it may be autoclaved) can be given subcutaneously. Adequate fluids should be given, parenterally if necessary. The patient should be kept in bed and away from sunlight, if possible. The urine should be kept alkaline with oral sodium bicarbonate or sodium lactate to prevent precipitation of sulfonamide crystals in the kidney tubules. Saline cathartics and sulfur-containing drugs, which hasten the development of sulfhemoglobinemia, should not be given during sulfanilamide therapy. The patient should be asked about previous reactions to sulfonamides. If the kidney function is poor or the hemoglobin is low, or if the patient is jaundiced or pregnant, sulfonamides should be given cautiously. At present, the cost of sulfathiazole is five times that of sulfanilamide; sulfapyridine and sulfaguanidine are thirteen times as expensive, and sulfadiazine twenty-nine times.

8. Diehl, H. S.: (a) The Treatment of the Common Cold, J. Indust. Hyg. 17:48-65 (March) 1935; (b) Medicinal Treatment of the Common Cold, J. A. M. A. 101:2042-2049 (Dec. 21) 1933.

9. Menefee, E. E., Jr. and Speed, J. A.: Treatment of Infections of the Respiratory Tract with Sulfonamides, North Carolina M. J. 2:611-618 (Nov.) 1941.

10. Chlorobutanol (chloretone) . . . . . 130 mg. (gr. ii)
Atropine or ephedrine sulfate . . . . . 130 mg. (gr. ii)
Glycerin . . . . . 15 cc. (oz. ½)
Water, q. s. ad. . . . . 30 cc. (oz. i)

11. Alcohol . . . . . 4 cc. (dr. i)
Hydrogen peroxide . . 4 cc. (dr. i)
Boric acid (saturated solution) . . . . . 16 cc. (dr. iv)
Water, q. s. ad. . . . . 30 cc. (oz. i)

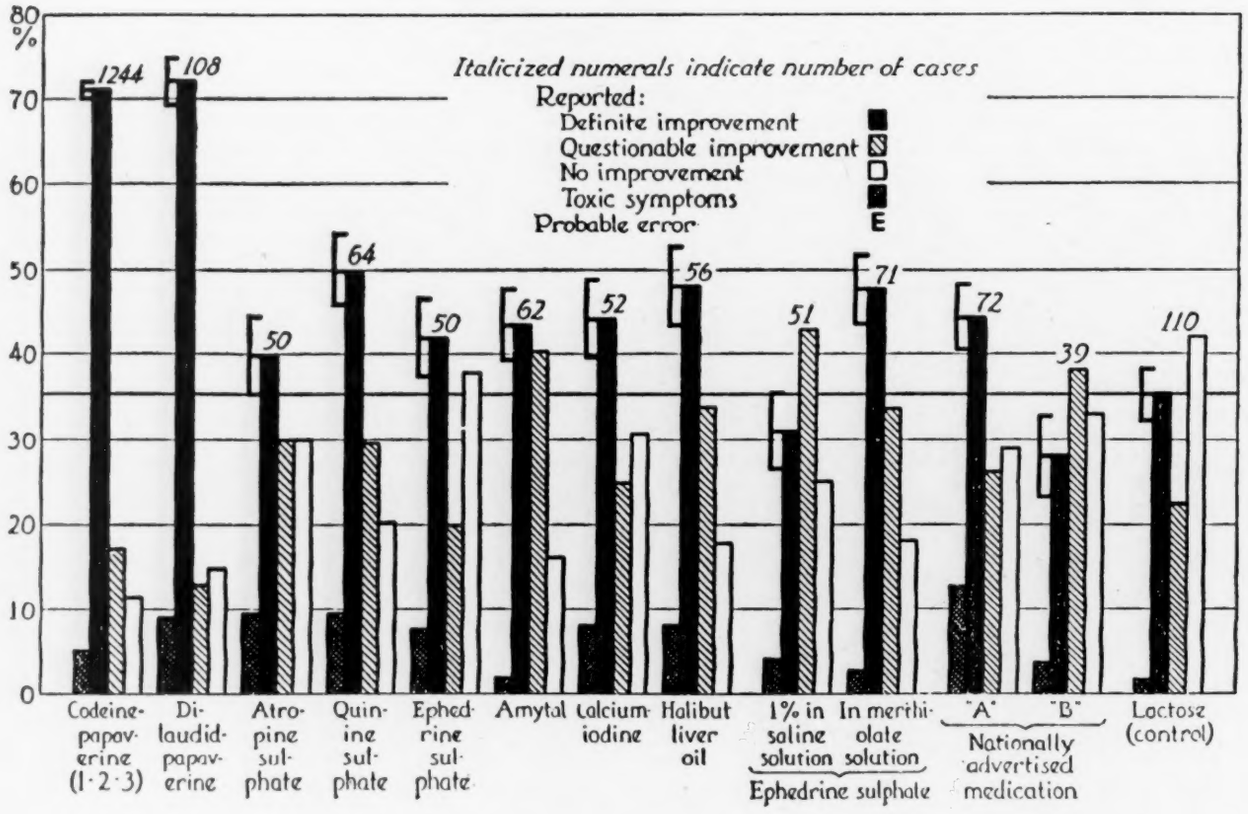

Fig. 1. Comparison of drug therapy in common colds (Courtesy H. S. Diehl, J. Indust. Hyg.[8], Williams and Wilkins Co.)

after it has been cleaned and dried, or by filling the canal with saturated urea solution or an extract of carica papaya, twice daily. Polyps, tonsils and adenoids should also be removed.

Mastoiditis: If the symptoms are definite and the mastoid cells are shown to be cloudy by x-ray, operation with thorough cleaning out of the cells should be done by a competent otolaryngologist. Oral sulfonamide therapy[7] is helpful.

Sinusitis: Nasal sprays of 0.25 per cent aqueous neosynephrin or 1 per cent aqueous racephedrine, Penetro or Benzedrine inhalers, and oral sulfonamide therapy[7] usually are sufficient. Relief from pain also may be obtained by dropping or spraying 1 per cent butyn into the nose four or five times at hourly intervals or by the ingestion of 0.3 Gm. (5 grains) of acetylsalicylic acid every four hours for three or four doses. Surgical interference rarely is necessary in children. Adenoids, hypertrophic or deviated turbinates, and other nasal obstructions may cause recurrent sinus infection and require removal while the infection is quiescent. If the sinusitis is due to allergy, the patient should be desensitized to the offending protein.

Cervical adenitis: Oral sulfonamide therapy[7] usually is sufficient.

Peritonsillar abscess: Oral sulfonamide therapy[7] and an ice collar occasionally will allay the inflammation, but if they do not, and the abscess is peritonsillar *and* fluctuant (an abscess never becomes fluctuant less than four days after onset), an incision should be made above the tonsil, with the patient's head *down* and *without* a general anesthetic, using suction to prevent the aspiration of pus. A tonsillectomy should not be done at the same time. If the abscess is retropharyngeal *and fluctuant*, it should be incised through the pharynx with a sharp fingernail or guarded scalpel, using suction to prevent the aspiration of pus.

Pertussis: Sedatives in small doses, especially at night, if the paroxysms are severe; raising the head of the bed; and the oral administration of 100-500 mg. of ascorbic acid daily are generally efficacious. Coughing usually is reduced while the patient is outdoors, or breathing moist air or steam. Two cubic centimeters of pertussis antigen (detoxified) every three days may shorten the course of the disease. Sulfonamide therapy[7] and oxygen are essential for

pneumonia. Three or four 20 cc. intramuscular doses, given every other day, of human "hyperimmune" pertussis serum (obtainable from the Philadelphia Serum Exchange, 1740 Bainbridge Street, Philadelphia, are life-saving in severe infections, especially if given early. An immune rabbit serum also is being used.

Bronchitis: Elixir of terpin hydrate or syrup of wild cherry with codeine should be added to the treatment for rhinopharyngitis discussed above.

Pneumonia: Sulfonamide therapy[7], small transfusions, an oxygen box[12, 13], and serum (if sulfonamide therapy fails) are the therapeutic measures available for pneumonia. Postural drainage and bronchoscopy may be necessary in chronic pneumonia.

12. Burgess, A. M., Briggs, A. S., and Burgess, A. M., Jr.: Oxygen by the Open Box Method, New England J. Med. 210:254-259 (Feb. 1) 1934.

13. A 35 to 65 per cent oxygen concentration can be maintained at the level of the patient's nose in a Burgess oxygen box[12]. The oxygen content can be raised to 95-98 per cent by covering the open top of this box with rubber sheeting or preferably Pliofilm no. 20 (Goodyear Rubber Co.) and hanging in the box a wire-mesh basket 12 x 2 x 2 inches, containing soda lime (which should be renewed every twenty hours) to remove carbon dioxide. If the box is open, soda lime is unnecessary, as the concentration of carbon dioxide is only 1 per cent with oxygen flowing at 2 liters per minute. The Burgess oxygen box is 20 x 20 x 18 inches; the sides and back are made of wood, and the bottom and front of the box are covered with Pliofilm no. 20 or canvas, tacked in place or fastened with thumb screws. This canvas should be removed and washed after each patient has used it. In the lower part of this front curtain is a 12 inch slit, closable with a "zipper", for admitting the head, thorax and arms of an infant, or the head of an older child. In use, the canvas floor is covered with a thin pillow. The wooden sides of the box contain windows of cleaned, non-inflammable, old x-ray films. A flattened metal gallon bucket is hung inside the box on a hook at the back. The space between the bottom of the bucket and the floor of the box should be 6 inches. This bucket should be kept filled with cracked ice and drained by rubber tubing leading from a metal tube soldered in the bottom of the bucket through the back of the box to a collecting pail on the floor. The condensed moisture on the outside of the ice bucket will not drip if a large rubber sponge is fastened with adhesive tape to the bottom of the bucket. A 10 x 10 inch piece of canvas or oilcloth, which acts as a baffle plate, is fastened vertically across the box flush with the top and parallel to and 5 inches in front of the back, separating the space for the patient's head from that occupied by the ice bucket. A 10 x 5 inch board is hinged at the top of the back to serve as a non-conducting cover to the ice cooler. An oxygen tank which delivers 2 to 3 liters a minute is connected to tubes which pierce one side of the box 3 inches from the bottom and 3 inches from the back. If it is necessary to elevate the patient's head, this box can be attached by metal clamps to the back of the bed. If desired, a larger box, closed on all four sides, can be used to contain an entire infant. The percentage of oxygen should be determined every four hours by means of an oxygen analyzer, which can be purchased or made. These boxes may be built locally or purchased from Technequipment Company, 199 Chapman Street, Canton, Massachusetts; Corp Brothers, 40 Mathewson Street, Providence, Rhode Island; or Warren E. Collins, 555 Huntington Avenue, Boston, Massachusetts.

Empyema: Drainage and Hart's tidal irrigation[14] should be employed in conjunction with sulfonamide therapy[7].

Tuberculosis: Pneumothorax and sanatorium care may be necessary.

Pleurisy: Paracentesis, sedatives, and diathermy may be employed in the treatment of pleurisy. In some cases sanatorium care may be necessary.

Foreign body: Bronchoscopy, plus sulfarsphenamine or sulfonamide therapy, is advisable.

Lung abscess and bronchiectasis: Sulfarsphenamine, sulfonamide therapy[7], postural and bronchoscopic drainage, potassium iodide, and lobectomy are the therapeutic measures available for these conditions.

Atelectasis and lung collapse: For newborn infants, the administration of 5 per cent carbon dioxide in oxygen, continuously or every half-hour, and gentle slapping every four hours to cause the infant to cry for ten minutes will usually expand the lungs. For older children, in addition to complete bedrest, similar carbon dioxide and oxygen therapy may be necessary. Any obstruction causing lung collapse should be removed bronchoscopically if possible. Postural drainage, expectorants, epinephrine, artificial pneumothorax, and desensitization may be necessary.

Pneumothorax: Bedrest is the only treatment.

Psittacosis and pleurodynia: Most of the patients recover without special therapy, other than sedatives. Convalescent immune serum shortens the course in psittacosis.

Fungus infections: If a skin test with the infecting fungus is positive, the patient should be desensitized, and *then* given a daily inhalation of 1 to 4 cc. (¼ to 1 drachm) of ethyl iodide by an inhaler, which can be

14. Hart, Deryl: Empyema in Children: Report of Thirty-Six Cases Treated by Tidal Irrigation and Suction, South. M. J. 23:823-829 (Sept.) 1930.

purchased from Warren E. Collins Company, Boston. A saturated (100 per cent) solution of potassium iodide also may be administered by mouth, starting with 0.1 cc. (1½ minims) three times a day, and increasing gradually to a daily total of 2 to 8 cc. (½ to 2 drachms), diluted with water. Oral and local sulfonamide therapy[7] is beneficial.

Hay fever and asthma: The acute symptoms may be relieved by the subcutaneous administration of 0.1 to 0.2 cc. (1 to 3 minims) of 1:1000 epinephrine every fifteen minutes for four doses, or by inhalations of 1:100 epinephrine in saline. The following preparations also relieve symptoms: 25 mg. (3/8 grains) of propadrine hydrochloride every three hours; aminophylline, ephedrine or amytal capsules[15]; 1 to 3 teaspoonfuls of 10 per cent potassium chloride or potassium citrate three times a day with or after food, *or* 0.3 to 1 Gm. (5 to 15 grains) of calcium phosphate (dibasic), calcium lactate, or calcium gluconate three times daily, *or* 0.3 to 0.6 Gm. (5 to 10 grains) of potassium chloride dissolved in a glass of water or milk three times a day. The maximal daily dose of the last preparation is 3 Gm. (45 grains), and it should be discontinued if nausea, diarrhea, epigastric pain, urinary frequency, or aggravation of asthma occurs. Helpful adjuncts to treatment are percomorph, cod or halibut liver oil, viosterol, ascorbic acid, orange or tomato juice, oxygen and helium therapy, air-conditioned rooms, a pollen-free climate, "slow epinephrine" (though it may cause reactions), nasal sprays of 0.25 per cent aqueous neo-synephrin or 1 per cent aqueous ephedrine, a benzedrine inhaler (not oftener than every hour, and not after 4 p.m.). For permanent cure, the causative proteins must be discovered by history or skin tests, and the patient must avoid them or be desensitized to them. Sinusitis and other sources of infection, and psychic factors should be sought and corrected. Tonsillectomy improves 50 per cent of asthmatic patients, but makes a few worse. Oral desensitizing preparations have not proved successful and may be dangerous. The pillows of children who are sensitive to feathers may be painted with liquid rubber, or covered with rubber pillow cases (obtainable from the Allergia Company, Newton, Mass.).

15. Aminophylline . . . 50 mg. (gr. 3/4)
Phenobarbital . . . 8 mg. (gr. 1/8)
Ephedrine sulfate . . 8 mg. (gr. 1/8)

A COMPARATIVE STUDY OF PREGNANCY IN THE WHITE AND COLORED RACES

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CHARLOTTE

The notoriously high maternal death rate in the South has been ascribed primarily to social and economic causes. A major factor is thought to be the large percentage of the child bearing populace among the colored race. The maternal mortality rate among Negroes throughout the country has always been higher than among the white population and has failed to show the substantial reduction that has been obtained in the mortality among white women during the past few years. In the United States the maternal mortality rate for Negroes in 1938 was 8.6 per 1000 births as compared with 9.3 in 1934—a reduction of 8 per cent. The mortality rate for white mothers in 1938 was 3.8 per 1000 births as compared with 5.4 in 1934—a reduction of 30 per cent. The maternal mortality rate among Negroes throughout the entire United States in 1938 was more than double the white mortality rate. North Carolina's statistics were comparable to those for the United States as a whole in this respect, the total maternal death rate in 1938 being 5.6 per 1000 live births: 4.4 for white women, 8.3 for Negroes.

This comparative study of 1000 consecutive pregnancies in indigent white women and 1000 in colored women, all residing in Mecklenburg County, was undertaken in an effort to ascertain if the colored patient is a greater obstetrical risk than the white patient living under similar social and economic circumstances. Private white patients naturally do not give fair comparative data. The 1000 white women herein reported were all indigent patients living in the poorer sections of the city or on tenant farms, and their dietary, economic and environmental circumstances are comparable to those of the Negro patients. Such studies as this, undertaken on a wide scale, might aid in answering some of the problems of the high mater-

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Appreciation is hereby expressed to Dr. Everett Sawyer for his aid in collecting the data summarized in this paper.

nal mortality in the Southern states. Is the rate in the South high because of the large proportion of Negroes, or is it high because of the social and economic conditions in which the Negro lives and breeds? In short, is race *per se* really a factor in maternal mortality?

CHART 1
Comparative Maternal Mortality by Race
North Carolina Bureau of Vital Stastistics

	Rate per 1000 live births	
	White	*Colored*
1930	6.7	12.2
1932	5.8	10.3
1934	6.7	9.7
1936	5.9	9.6
1938	4.5	8.4
1939	4.0	6.7
Total deaths 1930—640		
Total deaths 1939—394		

The Charlotte Maternity Clinic offers a prenatal service for indigent patients in a large central city clinic and five sub-clinics in rural sections of the county. This means that prenatal care is readily accessible to every resident of the city and county. In the early years of this program it was thought that the Negroes would be too indifferent and ignorant to avail themselves of this service. Such has not been the case; rather the white patient has proven to be less responsive and more immune to maternal welfare education than the Negro. One hundred and eight white patients in this series had no prenatal care, whereas there were only 21 Negroes who received none. Six hundred and eighty-nine Negroes had excellent prenatal care, as compared to 562 white patients. The illiterate white class is in certain respects a greater social problem than the Negro. Apparently they are less interested in receiving competent obstetrical care.

CHART 2
Prenatal Care

	1000 Colored	*1000 White*
None	2.1%	10.8%
Inadequate (4 visits or less)	29.0%	33.0%
Good	68.9%	56.2%

CHART 3
Age Group

	1000 Colored	*1000 White*
10-19 years	35.2%	20.5%
20-29 years	50 %	51.0%
30-39 years	14 %	25.0%
40 plus years	0.8%	4.5%

This analysis conforms with the accepted knowledge as to the early reproductive activity of the Negro and her declining fertility in the fourth and fifth decades of life.

CHART 4
Gravidity

	1000 Colored	*1000 White*
Primiparas	30%	22%
Multiparas	70%	78%

CHART 5
Hemorrhage

	1000 Colored	*1000 White*
Postpartum	21	23
Placenta praevia	5	3
Premature separation of the placenta	5	6
Total percentage	3.1%	3.2%

No racial difference is noted in the incidence of hemorrhage complicating pregnancy.

CHART 6
Labor

	1000 Colored	*1000 White*
Precipitate delivery	27.9%	14.2%
Labor over 24 hours	6.4%	6.6%
Labor over 48 hours	1.1%	1.5%

Aside from a higher incidence of precipitate delivery in the Negro (naturally high in any home delivery service) there is no striking racial difference in the duration of labor. The incidence of prolonged labor is quite similar in the two groups.

CHART 7
Operative incidence

	1000 Colored	*1000 White*
Low forceps	1.2%	3.7%
Mid forceps	0.4%	0.7%
Version	0.4%	0.1%
Breech extraction	0.2%	0.6%
Curettage	0	0.2%
Hysterotomy	0.6%	0.3%
Cesarean section	0.9%	1.0%
Total	3.7%	6.6%

The higher operative incidence in the white race is due chiefly to a greater use of low forceps in these patients (37 cases in the white patients as compared to 12 in the Negroes). Elimination of this factor leaves a similar incidence of major obstetrical procedures in the two races. Even the incidence of cesarean section shows no racial difference; there were 9 cases in the white group and 10 cases in the colored.

From the study of the duration of labor and the operative incidence it would seem that no pelvic racial variation occurs that is not well compensated by other factors. While contracted pelvis is generally accepted to be more frequent in the colored race, nature apparently terminates pregnancy as effectively as in the white patient.

CHART 8
Toxemia of Pregnancy

	1000 Colored	1000 White
Pre-eclampsia	11.4%*	6.9%†
Eclampsia	1.5%*	0.5%*
Hypertensive vascular disease	5.8%*	7.9%*
Total	18.7%	15.3%

* One maternal death.
† Two maternal deaths.

The incidence of eclamptogenic toxemia of pregnancy was found to be higher in the Negroes than in the white patients studied. This is the most positive finding in this study and this condition probably contributes to the higher Negro maternal mortality reported in vital statistics.

CHART 9
Induction of Labor

	1000 Colored	1000 White
Rupture of membranes	1.7%	2.8%
Bougie	0.1%	0.1%

CHART 10
Complications

	1000 Colored	1000 White
Organic heart disease	0.6%	0.6%
Tuberculosis	0.5%	0.3%
Fetal abnormalities	0.5%	1.4%
Syphilis	23.7%	5.9%
Puerperal infection	0.8%	1.9%

The high incidence of syphilis in the colored patients (237 cases in this series of 1000 deliveries), while probably not a factor in maternal mortality, requires comment as a contributing cause of maternal morbidity and fetal mortality. The higher incidence of fetal abnormalities in the white patients conforms with the published observations of Douglas Murphy on this subject. No other striking differences in the incidence of medical complications were noted.

An apparently higher incidence of infection in the white patients is not worthy of evaluation, for the delivery of the majority of these patients in the home did not permit an accurate study of morbidity.

CHART 11
Infant Mortality

	1000 Colored	1000 White
Miscarriages	2.4%	3.4%
Stillbirths	4.0%	3.3%
Neonatal deaths	4.2%	3.0%
Total infant mortality	10.6%	9.7%

No striking racial difference appears in the incidence of stillbirths. The incidence of miscarriages is similar. The higher incidence of neonatal deaths in the colored patients is probably related to the higher incidence of syphilis in this group.

CHART 12
Maternal Mortality

	White	Colored
Toxemia of pregnancy	2	4
Pneumonia	0	1
Postpartum hemorrhage	1	1
Ruptured uterus	1	0
Spinal anesthesia	1	0
Total	5	6

Case Notes of White Maternal Deaths

Case 1. This patient had hypertensive cardiovascular disease, and was first seen at the twenty-eighth week of gestation. Porro cesarean section was performed following premature separation of the placenta (Couvelaire uterus). Death occurred eleven days postoperatively.

Case 2. This patient had pre-eclamptic toxemia with acute cardiac decompensation. She received no prenatal care.

Case 3. This death was from eclampsia. The patient was first seen in labor with disproportion; forceps were unsuccessful; the patient died in shock.

Case 4. This patient had pre-eclampsia and disproportion. A low flap cesarean section was performed. Death resulted from infection. This case received adequate prenatal care.

Case 5. This was a case of bronchopneumonia and pneumococcic peritonitis. The patient received adequate care.

Case 6. This patient had mild pre-eclampsia, anemia, and an acute respiratory infection. Death was due to postpartum hemorrhage and shock.

Toxemia of pregnancy was a major factor in five out of the six maternal deaths.

Case Notes of Colored Maternal Deaths

Case 1. Death in this case was due to postpartum hemorrhage. The infant was born without a physician and the placenta was pulled out by the mother.

Case 2. This patient had a ruptured uterus. High forceps and a craniotomy were necessary to deliver a 12 pound infant.

Case 3. In this case death was due to eclampsia. One prenatal visit was made three months before delivery.

Case 4. This was the death which was due to spinal anesthesia. This case was complicated by cephalo-pelvic disproportion, pre-eclampsia, upper respiratory infection, and obesity.

Case 5. This patient had chronic hypertensive cardiovascular disease. The first visit was made when the patient was seven months pregnant. Her blood pressure was found to be 200 systolic, 140 diastolic. She was immediately admitted to the hospital, and death resulted from a sudden vascular accident at 6 a. m. the following day.

Toxemia of pregnancy was a major factor in three of the five colored maternal deaths.

From the standpoint of assignment of primary responsibility the maternal deaths occurring in this series of 2000 indigent patients may be divided into three arbitrary groups: those due to the patient, those due to the physician, and those non preventable.

CHART 13

Assignment of Major Responsibility for Maternal Deaths

	Colored	*White*
Unavoidable	0	2
Physician	2	2
Patient	3	2

Even in a fairly concentrated area of population where adequate maternal care and hospital facilities are provided the indigent patient frequently does not utilize such services. Often the patient's failure to cooperate intelligently results directly in a maternal death.

The very nature of a free clinic service results in limitation of professional interest and a lower professional standard than that represented in the relationship of qualified private physicians to paying patients. In addition to inadequate medical supervision the problem of a changing and inexperienced medical personnel—the inevitable price of a teaching service—is indisputably a factor in maternal mortality.

In the group of unpreventable maternal deaths the irreducible minimum has yet to be defined. The very nature of pregnancy, however, entails hazards that are occasionally fatal.

Conclusions

1. The incidence of toxemia of pregnancy and of syphilis is higher in the colored than in the white race.
2. No striking racial differences were noted in the incidence of hemorrhage, infection, operative deliveries, or other contributory causes of infant and maternal mortality.
3. Toxemia and chronic vascular pathology together constitute the outstanding factor in maternal mortality in the indigent, being a major contributing cause in eight of the eleven maternal deaths in this series of 2000 patients.
4. The Negro patient is often more appreciative, cooperative and receptive to an organized maternal welfare program than the indigent Southern white patient.
5. The high maternal mortality throughout the South is not primarily due to the large proportion of the colored race in the population, but to the social and economic circumstances confronting a large percentage of the populace, including the Negroes. Race *per se* is not a major contributing factor in maternal mortality.

Abstract of Discussion

Dr. Oren Moore (Charlotte): That paper is one of the most illuminating I have heard for a long time. We have for a great many generations used the large colored population as an alibi for the higher maternal death rate in the South. If the authors of this paper have done nothing else, they have taken away from us that alibi. This paper shows that the hazards of child bearing in the colored population are not a racial problem. This statistical study gives us other reasons for our high maternal death rate.

Four deaths out of 2000 cases are attributed to the physician. That factor ought to be one that we could eliminate. Doctors will continue to be human and fallible, and to lose some patients by errors in judgment. However, there ought not to be four deaths due to doctors in 2000 cases.

The incidence of toxemia is certainly too high, even in a clinic of this kind.

There was no death attributable to syphilis. It is amazing to see how many syphilitics are delivered in that clinic and what a small percentage of fetal mortality results from syphilis, although the patients get only an average of four or five doses of salvarsan during the prenatal period. There are very few difficult deliveries that can be attributed to syphilis.

A certain number of these patients are repeaters. Some of those who died with toxemia had probably been in the clinic with previous pregnancies and had been classified as toxemic. There is a point for birth control and sterilization. The paper is full of thought provoking material, and I am glad to have had this opportunity to discuss it.

Dr. A. L. deCamp (Fayetteville): My experience has borne out Dr. Bradford's conclusions that colored patients, on the whole, are far more receptive to prenatal care than are white patients. We have a small maternal clinic for colored women in Cumberland County which has gradually grown until we have to work long past hours to get through. At the white clinic, which is conducted on the same premises, we have a hard time getting enough patients to come to make it worth while to keep it open. The ratio of the white to the colored population in our county is about 6:4. We have that same type of white indigent patient who is very uncooperative and very indifferent to suggestions that are made about the management of pregnancy.

TREATMENT OF FLATULENCE WITH PROSTIGMIN BROMIDE

R. HENRY TEMPLE, M. D.

KINSTON

Even the layman is familiar with the symptoms of indigestion, and he has many labels for his discomfort: biliousness, heart burn, dyspepsia, sour stomach, gas attacks, and bloated feeling. Most often there is a disagreeable sensation in the epigastrium, often temporarily related to the taking of food and described as a burning, pressing, or full feeling. Usually the patient has difficulty in describing the sensation, as the symptoms change in their duration, intensity, and location from time to time. Sometimes it seems to flow upward over the front of the chest with burning or squeezing sensations in the throat. Oftentimes fullness and pressure cause pain around the left chest, with cardiac palpitation. Flatulence in one form or another is an almost universal complaint, and it is important that the physician be aware of this fact in order that he may properly evaluate the patient's story.

The causes of flatulence may be grouped as follows:

(1) Lesions in the stomach itself: carcinoma, achylia, peptic ulcer with hyperacidity and pylorospasm, gastritis, tumors of the stomach, phytobezoars, syphilis, deformities, and anomalies.

(2) Organic abdominal disease outside of the stomach, such as gallbladder disease, cardiac decompensation, chronic appendicitis, and coronary disease.

(3) Abnormal reflexes in the absence of any organic lesions, causing what is commonly known as nervous indigestion. The patient is usually the high strung type, and the actual symptoms are almost always directly related to worry, stress and strain, or to poor habits in eating, rest, and play[1].

It should always be the aim of the physician to approach the diagnosis of every case in which there is a complaint of flatulence by the purely eliminative process; for although it is undeniably true that the majority of digestive complaints are due to sensory, secretory, or motor disturbances and are therefore functional rather than organic in origin, yet the organic lesions which may cause digestive disturbances are so serious that it is absolutely essential that they be ruled out before a diagnosis of a purely functional dyspepsia is accepted[2].

Gas is normally found in the gastro-intestinal tract in small amounts. When the amount becomes excessive it produces a bloated feeling or sense of fullness, with the desire to expel gas orally or per rectum. This symptom of flatulence is probably the most common complaint that a physician encounters in taking a history; oftentimes it is the chief or sole complaint of the patient.

The physiology of the gaseous contents of the stomach and intestines takes one into a very intriguing field. Most gas that occurs in the stomach is swallowed and consists chiefly of atmospheric nitrogen, of which practically none is absorbed either in the stomach or in the intestines. The remaining gaseous substance in the stomach is oxygen, which is partially absorbed along the gastro-intestinal tract. The hydrochloric acid content of the stomach prevents fermentation and the formation of additional gas in the stomach. Usually the greatest amount of gases are formed in the cecum and ascending colon, including the hepatic flexure. This fact is partially explained by the large quantity of liquids associated with stasis, and the increased bacterial flora coming into contact with the intestinal contents. The gases found in the intestines are carbon dioxide (which is mostly absorbed by the blood and expired through the lungs, except in certain pathological conditions), methane, hydrogen, indole, skatole, ammonia, and hydrogen sulfide. The nitrogen traverses the entire intestinal tract and is expelled as flatus with other gases[3].

Flatulence *per se* may be due to excessive intake, as in aerophagia—a condition which is difficult to explain to patients; many are convinced only after fluoroscopic demonstration. Excessive formation of gas accounts for many cases and results from incomplete decomposition due to an abnormal intestinal flora or a diet excessive in protein, carbohydrates, or cellulose. Oftentimes the incomplete digestion of starches, owing to insufficient cooking or poor mastication, may cause excessive gas. Some cases are due to deficient expulsion of gas as the result of constipation, redundance of the colon, ob-

Read before the Regional Meeting of the American College of Physicians, Chapel Hill, October 31, 1941.

1. Musser, J. H.: Internal Medicine, ed. 2, Philadelphia, Lea and Febiger, 1934, p. 594.
2. Beckman, Harry: Treatment in General Practice, ed. 2, Philadelphia, W. B. Saunders, 1934, p. 403.
3. Boles, Russell S.: Personal communication.

struction from a spastic sphincter ani, or a voluntary restraint of gas in the bowel (which is not at all an uncommon cause of flatulence).

The deficient absorption of gas from the intestinal tract due to a circulatory disturbance in the bowel and stomach (for example, a failing heart, hypertensive cardio-renal disease, coronary artery disease, nephritis, cirrhosis of the liver, hepatitis, and cancer of the stomach), or to an impairment of the absorptive powers of the mucosa in gastroenteritis or colitis may be the factor responsible for excessive gas[4].

Intestinal atony is a common cause of flatulence, and it may be neurogenic or toxic in origin. Common examples of this condition are found in cases of hysteria, neurasthenia, typhoid fever, and pneumonia, all of which may retard gas absorption and promote increased formation[5].

Anemia may be a much more important factor than it is supposed to be. It was present in a majority of the cases reported in this paper. The theory is that the gas carrying properties of the blood (chiefly the hemoglobin and mature erythrocytes) are deficient, and the transportation of gas from the intestines to the lungs is impeded. Respiratory diseases such as lobar pneumonia, asthma, and pulmonary emphysema may increase the intestinal gas because of the difficulty in ridding the blood of waste gas by respiration[6].

Treatment

The treatment of flatulence should, of course, be directed to the underlying cause. Extreme degrees of tympanites associated with colic require immediate palliative treatment. In such cases reported in this paper in which adhesions were causing a partial intestinal obstruction[7], the usual treatment of the underlying pathology was instigated, with varying results; then prostigmin bromide tablets were used after a period of three or four weeks.

As flatulence often has a neurogenic basis, the following treatment of symptoms was followed in cases of persistent flatulence in which intestinal studies along with x-rays revealed no organic lesion.

(1) Sedation (This is of paramount importance.)
(2) Psychotherapy
(3) Rest
(4) Hot fomentations
(5) Belladonna, benzedrine, calcium gluconate
(6) Carminatives: spirits of peppermint, Hoffmann's anodyne
(7) Correction of aerophagia, if present
(8) Correction of constipation
(9) Diet and vitamin B complex[8]
(10) Exercise

After three or four weeks, whether the results were good or poor, the patients were put on prostigmin bromide, 15 mg. four times a day. This was given in all cases, regardless of the pathology.

Chemistry and Pharmacology

Prostigmin bromide is the dimethyl-carbamic ester of m-oxyphenyltrimethyl ammonium bromide.

In general, it may be said that the pharmacological action of prostigmin is similar to that of physostigmine (eserine), with the important distinction that the prostigmin is practically free of the disturbing effects which attend the use of physostigmine. Prostigmin inhibits the destruction of acetylcholine at the myoneural junction by combining the urethane groups of the prostigmin molecule with the "esterase", thereby retarding its destructive action on acetylcholine and hastening the stimuli from the motor nerves to the voluntary muscles[9].

However, the most important property of the drug in the treatment and prevention of gas in the intestinal tract is its activity as a vagotonic agent. It causes a marked increase in peristalsis and stimulates an atonic intestinal tract. All the muscular activities of the small intestines are stimulated by prostigmin. Symptoms of overdosage are muscular twitchings, weakness, vertigo, intestinal hyperperistalsis, and repeated bowel movements. Atropine or belladonna are the best antagonists to prostigmin[10].

Prostigmin has a mild miotic effect, and causes a very slight change in blood pressure[11]. It restores the tonicity of the

4. The Cyclopedia of Medicine, Surgery, and Specialties, Philadelphia, F. A. Davis, 1939, vol. 6, p. 326.
5. Kantor, J. L., and Marks, J. A.: Study of Intestinal Flatulence, Ann. Int. Med. 3:403 (November) 1929.
6. Alvarez, W. C.: The Mechanics of the Digestive Tract, ed. 2, New York, Paul B. Hoeber, 1928, p. 347.
7. Thewlis, M. W.: Preclinical Medicine, Baltimore, Williams and Wilkins, 1939, p. 74.
8. Haggard and Greenburg: Diet and Physical Efficiency, New Haven, Yale University Press, 1935.
9. The Cyclopedia of Medicine, Surgery, and Specialties, Philadelphia, F. A. Davis, 1940, vol. 12, p. 632.
10. Written communication from Hoffman-LaRoche, Inc., Nutley, N. J., September 4, 1941.
11. Shaine, M. S.: The Treatment of Flatulence, Rev. Gastroenterol. 8:181 (March and April) 1941.

intestines to slightly more than the normal level[12].

Results Obtained With Prostigmin Bromide

In February, 1940, a patient suffering with a nephroma was given 30 mg. of prostigmin bromide at bedtime and 30 mg. in the morning preparatory to intravenous urography[13]. The radiographs were excellent, with very few gas shadows. During the examination the patient inquired about the tablets and remarked that she had expelled much gas and felt greatly relieved. Flatulence had been a prominent symptom in her case. The patient was given 15 mg. of prostigmin after each meal until her death in August of the same year. I felt that this unfortunate patient had been given great relief from her abdominal symptoms by the use of prostigmin orally and decided to use it symptomatically in the treatment of all patients complaining of flatulence, regardless of the cause.

The appearance of distention due to toxic conditions such as typhoid fever and pneumonia[14], or to abdominal or pelvic adhesions causing a partial obstruction is a warning that the normal tonicity of the intestinal mucosa has been lost. When the motility of the gastric and intestinal mucosa has been impaired, the resulting atony is often followed by markedly increased secretion from the walls of the stomach and intestines. As absorption from the gut decreases, the bowels become filled with large quantities of toxic fluid. As a result of the increased pressure, the blood vessels within the intestinal wall are compressed. This causes a complete cessation of peristalsis and results in acute gastric dilatation and paralytic ileus[15]. I feel that, in 2 of the cases reported in which there was partial obstruction, the prostigmin accounted for the unusually smooth postoperative convalescence[16].

12. Klingman, W. O.: The Treatment of Neurogenic Megacolon with Selective Drugs, J. Pediat. 13:805 (December) 1938.
13. Marden, P. A. and Williamson, E. G.: Use of Prostigmin Methylsulfate in Prevention of Post-Operative Intestinal Atony and Urinary Retention, Surg. Gynec. & Obst. 69:61 (July) 1939.
14. Tractenberg, I., and Oliver, W.: The Use of Prostigmin in Abdominal and Vaginal Operations for Relief of Postoperative Distention and Urinary Retention, 53:284 (August) 1941.
15. Harger, J. R. and Wilkey, J. L.: Management of Post-Operative Distention and Ileus, J. A. M. A. 110:1165 (April 9) 1938.
16. Levis, W. R. and Axelman, E. L.: Modern Method for Prevention of Post-Operative Distention, Report of Eighty-Eight Cases, Am. J. Surg. 32:308 (May) 1936.

I have treated 29 cases of functional flatulence with prostigmin bromide. The ages range from 20 to 57 years. Many of these patients had minor disorders that were corrected as part of the treatment. After they had been under a general routine of treatment for three weeks or more, an attempt was made to evaluate their improvement. All felt that they had improved to some extent. However, every patient still suffered from flatulence either continuously or at intervals. Each of these patients was then given prostigmin bromide tablets, 15 mg. each, three to four times daily. In every instance great relief was afforded. Patients who suffered from gas only at intervals were given medication only during the attacks.

Eighteen cases of gastric anacidity have been treated by this method. In 2 cases syphilis was the etiologic factor, and in one case bronchogenic carcinoma was responsible for the anacidity. All of the patients were given glutamic acid and vitamin B_1, with most gratifying results, except in one case. However, virtually every patient stated that he was still extremely bloated and very uncomfortable one or two days during a week. Prostigmin bromide was prescribed, with instructions to use it orally as needed. Every patient seemed greatly helped except the one who failed to notice any benefit from the glutamic acid.

Four cases of partial intestinal obstruction from pelvic and abdominal adhesions have received prostigmin bromide. One of the cases was syphilitic. Each of the 4 patients obtained temporary relief from distention and tympanites with prostigmin medication, used in conjunction with a rectal tube and hot fomentations. In 2 cases operations were performed to relieve the obstruction. One patient with multiple fibroids will be operated on soon.

I have treated 7 cases of gallbladder disease, in 2 of which there was cholelithiasis, by this method. Routine symptomatic treatment was given for cholecystitis with fair results. Prostigmin bromide orally relieved the gas and bloating, but in no instance was helpful in alleviating the pain.

Eight patients with hypertensive cardiorenal disease marked by much gas and bloating were given prostigmin bromide. One of the patients suffered a coronary occlusion and was extremely uncomfortable for three days because of the tremendous quantities of gas that accumulated in his alimentary

canal. Prostigmin medication in this instance had almost immediate effect. The other 7 patients received some relief from prostigmin, but results varied a great deal.

Summary and Conclusions

Prostigmin bromide in 15 mg. tablets, given orally three or four times a day, relieved the flatulence in 64 patients: 29 with functional flatulence and minor disorders, 17 with gastric anacidity, 4 with abdominal and pelvic adhesions, 7 with gallbladder disease, and 7 with hypertensive cardiorenal disease. Two patients—one having gastric anacidity, anemia, and hypertension with mild cardiorenal disease, and the other with angina pectoris, cirrhosis of the liver, and anacidity —received no benefit. No untoward reactions were encountered in the 66 cases reported.

This series of cases is, of course, insufficient to warrant the indiscriminate use of prostigmin in all cases of flatulence. Some cases reported were not greatly benefited; however, it seems that the results would indicate that the use of prostigmin should not be restricted to cases of purely functional flatulence. The only caution to be strictly observed is that the drug should not be used in cases of complete intestinal obstruction.

Education for Old Age — Education, which is preparation, has not kept pace with these changes in the social order. Educational curriculums are still geared to the day when life expectancy was fifteen or twenty years less than it is today, when it sufficed that education prepare the boy or girl for the competition of adult life. Neither parents nor teachers have taken cognizance of the necessity for preparation for old age. It has been assumed with complacent smugness that the adult would learn how to grow old—gracefully, happily and usefully—without training or aid. Unfortunately, very few learn this spontaneously. The time has come when educators must revise their objectives. The accomplishments of such men as Oliver Wendell Holmes, William H. Welch, Goethe, Edison, Titian and many others in the evening of their lives are mere indicators of the vast storehouse of latent treasure in those so often sneeringly dubbed "old men." Such careers are rarely fortuitous. Continued development is greatly enhanced by cultivation. — Edward J. Stieglitz: The Potentialities of Preventive Geriatrics, New England J. Med. 225: 248 (August 14) 1941.

Confidence.—When a patient goes to a physician he usually has confidence that the physician is the best, or at least the best available, person to help him in what is, for the time being, his most important trouble. He relies on him as on a sympathetic adviser and a wise professional counsellor.—Francis W. Peabody: Doctor and Patient, New York, The Macmillan Co., 1939.

THE FAMILY PHYSICIAN AND THE OCULIST

W. P. SPEAS, M. D.

WINSTON-SALEM

At the request of our secretary I am bringing you today some observations gleaned from the experience of thirty years of medical practice. The temptation is to reminisce, to talk of those times when we were in general practice doing everything from tracheotomies on the dining room table by a small kerosene lamp to arm presentation deliveries, when help of any kind was out of the question. Those were hard but happy days. As family physician we were looked up to. As one who has fallen from that high estate into the position of one who merely tries to cover one phase of medical practice, I feel that at least I am able to appreciate the position and problems of the family physician.

The so-called "specialist", no matter what his field, should never forget his position with regard to the whole profession. He should remember always that his specialty is only a branch of the parent trunk. He is still bound by the high standards of medical ethics that apply to the general practitioner. His responsibility in this respect is even greater, because he examines and treats patients of the general practitioner in the privacy of his office without the presence of the referring physician. The high regard in which the patient holds his family doctor must be handled as a sacred trust, so that in every case the patient goes back to his physician with his confidence unshaken, whether he has been referred by that physician or has come of his own accord.

Just when a case should be referred by the family doctor to the oculist is a decision often difficult to make. Certainly no hard and fast rule can be made in most types of cases.

Inflammations of the Conjunctiva

Most types of conjunctivitis can be treated by the family physician. The question whether a red eye is simply conjunctivitis is frequently difficult to decide, even for one trained in eye work. If the condition is of only a few days' duration, with no severe

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pain, and with normal pupillary reaction to light, the physician is usually safe in prescribing an antiseptic solution, rest, protection from light, and the usual general measures.

If, however, there is severe pain and a contracted or irregular pupil, one should suspect iritis. Especially is this true if the pain is worse at night. In case the pupil is dilated and does not react to light, and there is severe pain, one should suspect acute glaucoma. It would probably be unwise for the family physician to take the responsibility of treating these rather serious conditions.

Ophthalmia neonatorum, or purulent conjunctivitis in the new born, is still seen occasionally. These cases must be treated energetically, and most physicians do not care to take the responsibility.

Headache

Headache is a symptom often encountered in general practice. Here, too, it is often difficult for the conscientious physician to decide upon the proper course of action. The type of headache, and the occupation, age, and physical condition of the patient are important clues to the diagnosis. Paroxysmal attacks of headache, occurring every few days or weeks without any special relation to close work suggest some constitutional condition such as toxemia, or perhaps a familial predisposition. The headaches that occur early in the morning, especially if the eyes are not subjected to excessive work at night, do not usually come from the eyes. Rather a gastro-intestinal basis or sinus involvement should be suspected. These cases should be studied carefully before being referred to an oculist. If, however, headache comes on during the day's work, especially in students, bookkeepers, and clerical workers, one should be suspicious of eye strain.

Headache rarely occurs as a result of eye strain in people past middle life. After one becomes presbyopic the eyes give up as a bad job seeing keenly, especially in near work. The patient is thus spared the loss of nervous energy which had to be expended in the act of accommodation earlier in life. In older patients, the physician should search for some general cause of headache such as high blood pressure or gastro-intestinal toxemia. In cases of persistent headache, he should not forget the possibility of increased intracranial pressure. In such cases the oculist may help in making the diagnosis. In children and adults up to the presbyopic age, headache accompanying or following the use of the eyes in close work should always suggest eye strain.

Defective Vision

Every physician should have in his office a chart of the Snellen type with proper illumination for testing the vision of patients. All students and others engaged in close work whose vision is defective should be referred for refraction. In farmers and other patients whose occupation does not make exacting requirements for clear vision, vision as low as 20/40 is not objectionable, provided the patient is comfortable.

Eye Injuries

Injured eyes should be handled with extreme care. All patients with perforating wounds of the eye should be referred to the oculist. Here the danger of a later-developing sympathetic ophthalmia offers a medico-legal problem which might cause the practitioner much anxiety and embarrassment. A contused eye in which the vision is unaffected and the media are clear may be handled safely by the family physician.

Foreign Bodies in the Eye

The family physician should remove all loose or lightly attached foreign bodies, using 2 per cent butyn or 4 per cent cocaine as an anesthetic. It is not wise for the physician to prolong the effort to remove a foreign body from the cornea if it is found adherent. Nor is it wise for him to attempt to remove embedded foreign bodies, because of the danger of ulceration and possible impairment of vision.

Squint

I beg the indulgence of this society in mentioning again a subject which I brought to your attention at a previous meeting some five or six years ago—namely, the condition commonly known as squint or cross-eye. My only reason for giving the matter more than passing notice at this time is the fact that a large percentage of these cases do not get attention until it is too late to obtain more than partial relief.

"Crossed eyes" or "squint" usually becomes manifest in the pre-school age. Per-

haps the child has had some weakening sickness which the parents feel has "settled in his eyes". Or it may be noticeable only when the child is tired or embarrassed, as in the presence of strangers. Often beginning as an occasional condition, the squint usually becomes constant if left alone. The child grows as other children and is happy in his complete absence of self-consciousness. Often some well-meaning friend advises the parents to wait until the child is old enough to go to school before seeking relief. So, perhaps glasses are purchased for him when he is 6 or 7 years of age. If the treatment begins and ends here, the child will continue to squint ninety-nine times in a hundred. The treatment has been started too late and has ended too soon.

As time goes on the child becomes conscious of the fact that his crossed eyes make him conspicuous; he becomes sensitive about his deformity, and often avoids the company of playmates who sometimes taunt him in regard to his eyes. He thus develops an inferiority complex, which, together with his crossed eyes, is a handicap throughout life.

When we see a case of squint, we should think not only of the deviation of the eye from parallelism, but also of three less obvious, but none the less important conditions: (1) A defective fusion faculty, (2) poor vision in the eye that is out of line, and (3) some form of eye strain.

Fusion: By fusion is meant that faculty of the brain which causes the images in both eyes to merge into one. The normal individual looks with both eyes at an object. An image of that object is produced on the retina of each eye, so that there are two images of the same object. These are conveyed along the optic nerves to the brain and fused into one. For this to be accomplished by the brain, however, it is imperative that the eyes be perfectly straight. In the average individual this desire for binocular vision, this sense of fusion, is so strong that it holds the eyes in line even though they may have a tendency to converge or otherwise get out of line. If this fusion faculty is weak in a child. there is little to hold the eyes in line, and he becomes cross-eyed. The most unfortunate aspect of the situation is the early age at which this sense must be developed if it is to be developed at all. In the normal child it is fairly well developed by the time he is 1 year old, and it can be developed after the age of 6 or 7 only in rare instances. In a small percentage of children the fusion faculty is entirely absent and no amout of effort can develop it. These children with complete absence of fusion usually see equally well with each eye, crossing first one eye and then the other.

Poor Vision in One Eye: In the examination of a child, one of whose eyes has been crossed for a short while, it will usually be found that the vision of the squinting eye is defective. If the cross-eyed child were to see clearly with both eyes, all objects looked at would appear double. Nature abhors a double image, so she suppresses the image in the deviating eye. The younger the child, the more rapidly and completely does the eye lose the ability to see. A child a year old or younger may lose almost completely the power of fixation within two or three months. In children somewhat older the process is slower, but no less certain to destroy the ocular function if unimpeded.

Eye Strain: In addition to poor fusion and defective vision in the squinting eye, most cross-eyed children have a high refractive error, such as farsightedness. It is imperative that this be corrected at the earliest possible date. In cases of occasional squint it often happens that properly fitted lenses alone will correct the condition.

I have discussed this subject in detail in an effort to impress on the family physician the importance of referring these cases to an oculist. The family physician should make it a rule to refer them as soon as they are discovered, and there should be no exceptions to the rule. The responsibility will then be with the oculist. He will advise the parents as to the proper course of action in each individual case.

Conclusion

The family physician and the oculist should work at all times in closest harmony. In all our efforts we have a common end in view—namely, the interest and well being of the patient.

Gerontology — Gerontology is no longer merely academically interesting, but has become an urgent matter in the minds of those sufficiently far-sighted to see the handwriting on the wall. The nation is aging rapidly. The virile, violent but short-lived days of physical pioneering are largely past. The future holds promise of profound change. A period of intellectual conquest may be dawning. Man at last lives long enough to have time to think.—Edward J. Stieglitz: The Potentialities of Preventive Geriatrics, New England J. Med. 225:248 (August 14) 1941.

ENLARGED PARIETAL FORAMINA

KENNETH B. GEDDIE, M. D.

HIGH POINT

The condition of enlarged parietal foramina has been known to exist at least since Lancisi described it in 1707. Lobstein in 1772 again briefly described a skull specimen demonstrating this anomaly. Turner in 1865 gave the first succinct description of this condition: "At the commencement of the posterior slope of each parietal bone, an oval opening, possessing a rounded margin, was situated, the inner end of each of which was about half an inch from the middle line. The long axis of each opening was transverse, that on the right side measured 8/10th of an inch, that on the left 6/10th, the antero-posterior diameter of the right was at its widest 6/10th, while that of the left was only 4/10ths of an inch." In anatomical specimens studied this anomaly has been found in prehistoric races, in Egyptian mummies, and in various aboriginal races as well as in civilized man. The hereditary phenomenon which I am discussing today is something entirely different from the millimeter-sized parietal openings which occur not infrequently in normal adults. Pendergrass and Pepper recently described the normal development of skull ossification as follows[1]: "The normal ossification of the parietal bone commences in a single center at the end of the second fetal month. As the bones of the skull develop, there are six places where well-defined fontanelles exist. These are situated at the four angles of the parietal bones, two in the median line and two laterally on each side. In addition, there is present during fetal life in each parietal an interval between the radiating lines of newly formed bone along which ossification of the parietal progresses. This space is named the sagittal fontanelle. As the development of the parietal advances the sagittal fontanelle is encroached upon until, as a rule, about the seventh month of fetal life the fontanelle as such has disappeared and only the two minute parietal foramina remain open. Even in skulls which become wholly normal this process may be delayed and the sagittal fontanelle remain open until birth." The hereditary anomaly which we are discussing is due to an even greater delay in the process and to incomplete ossification of this region of the parietal bones.

The first case to be diagnosed during life was reported by Greig in 1892. The patient, one Robert Ross, aged 35, a driver in the Royal Field Artillery on duty in India, came under the observation of Dr. Greig in 1889 when he was referred to him for examination because he used the presence of two "soft spots in his head" as an excuse for some slight dereliction of duty. He later told Greig that these were congenital in origin and admitted that they gave him no trouble whatsoever. These openings Greig described as "a symmetrical deficiency at the posterior-superior angle of each parietal bone, the two openings being separated by a median bridge presumably corresponding to the Sagittal Suture." In 1917 Greig reported another case[2], with an x-ray picture of the condition. This patient was the brother of the case reported in 1892. In 1917 Greig could find reports of only fourteen examples of this deformity. In 1919 Pamperl recorded a case diagnosed by x-ray examination and collected 35 other cases from the literature[3]. At that time it was evident that the condition would be more and more frequently recognized as x-ray examinations became more frequent. This has been found to be true. There have been seven reports of studies of families in which several members of the family have been affected. Three other case reports mention the examination of some other members of the family but do not give a complete familial study. In 1922 Goldsmith[4] reported an important study of the familial occurrence of enlarged parietal foramina. This study of five generations of an American family (the Catlin family) caused him to conclude that: "The hereditary defect did not seem to fall in line with the usual laws of inheritance." His patient was of the third generation. The "mark" was traced upward to the father and grandfather (first and second generation). In the third generation four out of sixteen members studied showed the anomaly. Of twenty-eight members of the fourth and thirty-two members of the fifth generation studied,

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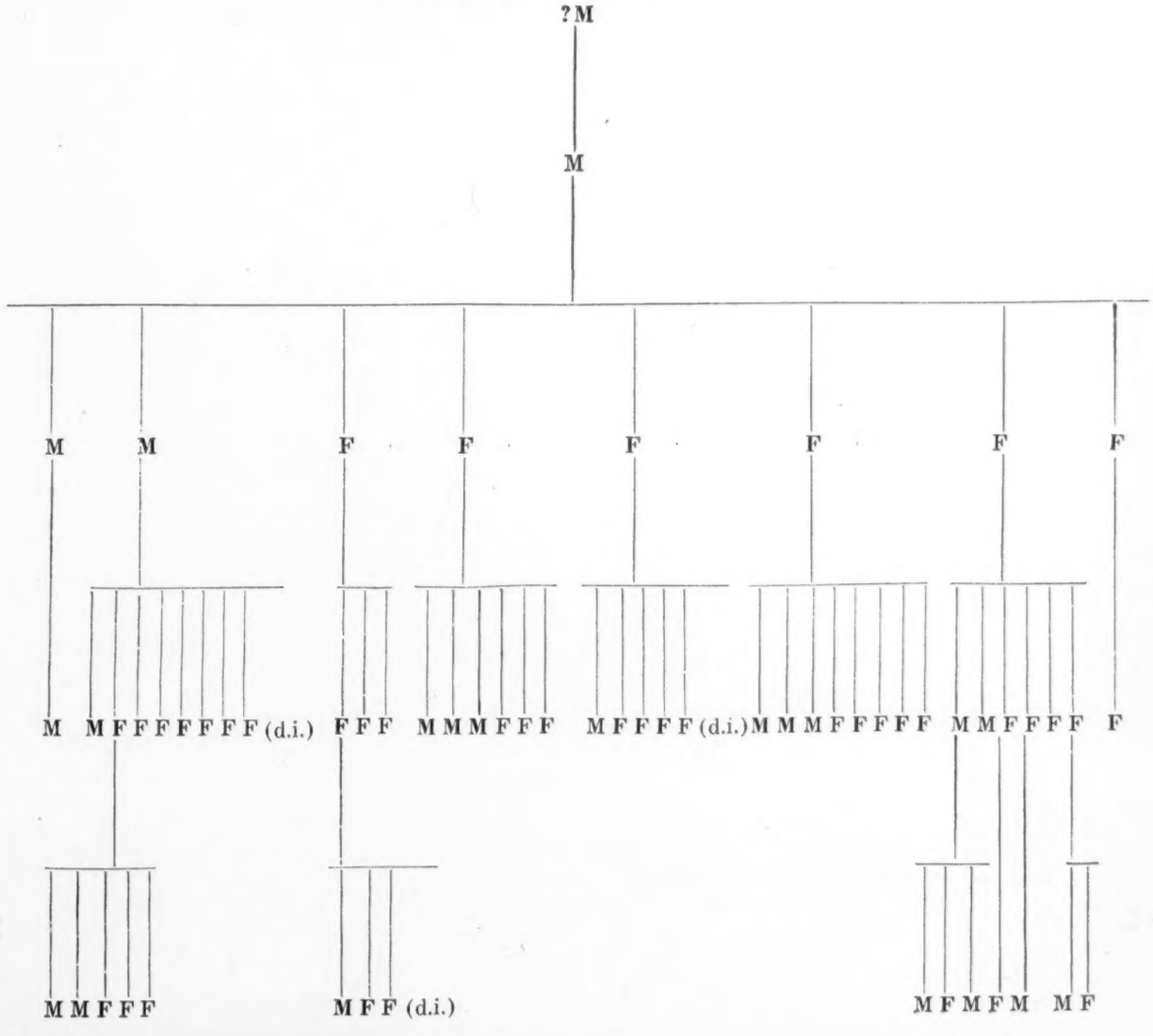
1. Pendergrass, E. P., and Pepper, O. H. P.: Observations on Process of Ossification in the Formation of Persistent Enlarged Parietal Foramina, Am. J. Roentgenol. 41:343 (March) 1939.
2. Greig, D. M.: Perforation of Parietal Bones, Edinburgh M. J. 18:205 (March) 1917.
3. Pamperl, R.: Abnormally Large Foramina in the Skull, Deutsche. Ztschr. f. Chir. 148:91 (January) 1919.
4. Goldsmith, W. M.: The Catlin Mark, J. Heredity 13:69, 1922.

ten were found to have this defect. One member of a pair of twins was affected. In 1924, Cohn reported this anomaly in both of a pair of twins (female) and in their mother[5]. In 1935 Pepper and Pendergrass presented a report on the hereditary occurrence of enlarged parietal foramina[6]. Of nine members representing four generations of a family five had definite enlarged parietal foramina and two showed bilateral foramina of smaller size but larger than normal. In the same paper these authors presented a partial study of a Negro family which showed three members to be affected. Other reports have appeared in the literature since this.

Many other anomalies and developmental defects have been reported in conjunction with this condition. Recently Warkany and Weaver reported "enlarged parietal foramina combined with obesity, hypogenitalism, microphthalmos and mental retardation."[7] They briefly reviewed the literature and presented many examples to indicate the frequency with which morphologic and functional abnormalities are seen combined with

5. Cohn, Michael: Hereditary Defects in Parietal Bone, Med. Klinik. 20:857 (June 22) 1924.
6. Pepper, O. H. P., and Pendergrass, E. P.: Hereditary Occurrence of Enlarged Parietal Foramina, Am. J. Roentgenol. 35:1 (January) 1936.
7. Warkany, J., and Weaver, T. S.: Heredofamileal Deviations, Enlarged Parietal Foramens Combined With Obesity, Hypogenitalism, Microphthalmos and Mental Retardation, Am. J. Dis. Child. 60:1147 (November) 1940.

PEDIGREE OF THE McKENZIE FAMILY, SHOWING OCCURRENCE OF ENLARGED PARIETAL FORAMINA



Letters in boldface type denote members affected.
d. i. denotes members who died in infancy.

enlarged parietal foramina. Other reports have stressed the familial and hereditary tendency and the absence of other developmental defects.

My study of the McKenzie family is the fourth complete familial study of this condition which I have been able to find recorded in the literature. It reveals the presence of this anomaly certainly in four generations and by hearsay in the fifth generation. In this family no other morphologic or functional abnormalities were found to be present. I personally examined each member reported and found the lesion on routine examination by inspection or palpation or both. Some of the larger lesions were pictured by x-ray, but x-ray examinations were not done on all the members. Doubtless other cases reported as negative would show the lesion if examined roentgenologically. No symptoms, not relative to the anomaly, were present in any individual. The lesions were of all variety of shapes and sizes. Most of them were irregularly angular, but some were symmetrically round. The largest opening occurred in the second generation and measured 10.2 by 4.7 cm. The x-ray report made by Dr. P. W. Flagge is as follows: "This is a very large defect, again showing a complete lack of bone in the sagittal line, except for a spur about 1.5 cm. in length on the lower border. The aperture measures 4.7 cm. by 10.2 cm. in the greatest diameter." The smallest lesion recorded was about 1 cm. in diameter. The youngest member examined was 8 months of age. The oldest member was 83 years of age. All are living. Two children in the third and one in the fourth generation had died. Whether or not the anomaly was present in these three children is not known.

The first patient that I saw who presented this anomaly was a girl 2½ years of age. She was of the fourth generation that I was able to check. When she was 5½ years of age the openings were of moderate size (3 x 1 cm. and 1.8 x .8 cm.). Although I did not measure the openings when I first saw her at 2½ years of age my impression is that they had decreased considerably in size in the intervening three years. Her father is the individual referred to above as having the largest opening in the whole group. Her grandfather had an unusually large opening but I do not have the exact measurements. Her great grandfather, aged 83, also had a bilateral opening. He stated that his father died when he himself was quite a child, but he thought that he too presented what he called the "McKenzie soft head".

Of eight persons in the second generation six had bilateral foramina. Of thirty-six persons in the third generation, fourteen had bilateral foramina and three had single openings. Of sixteen persons in the fourth generation three had bilateral openings and one a single parietal opening. Only one pair of twins were in this group, and one of these was affected.

Summary

Sixty-one persons in four generations of one family are described. Twenty-eight presented enlarged parietal foramina. The youngest was 8 months of age. The oldest was 83 years of age. No other anomalies or developmental defects were present in any member of this family.

Comment

Although I have read practically all the reported cases of this condition I have made no attempt to give a complete bibliography because that was done by F. J. Halbertsma in June, 1940[8], and by Warkany and Weaver in November, 1940[7]. This condition is probably not uncommon. It is primarily of interest because of its hereditary and familial incidence.

This condition is of no clinical importance except in the differential diagnosis of possible pathological conditions.

8. Halbertsma, T.: Fenestrae Parietales Symmetricae, Arch. Dis. Childhood 15:115 (June) 1940.

The detrimental effect of war on the morbidity and mortality of tuberculosis is well known from the lesson of the Great War of 1914-1918. According to the consensus of opinion it was due to a multitude of factors and circumstances, such as malnutrition, overcrowding, overwork and, in some countries, suspension or curtailing of public health services, diminution of beds available for treatment, etc. These conditions and circumstances cannot be eliminated from war and a similar increase in mortality and morbidity of tuberculosis is generally expected. In England there are at least five additional major factors present at this time which did not exist in the last war: intensive air raids, blackout, mass migration of the population, shelter life and conscription of women. Closer scrutiny of the tuberculosis problem, however, raises some hope for a more optimistic view. In 1914-18 the diagnosis of early tuberculosis was a difficult one, tuberculosis welfare was undeveloped, collapse therapy was restricted to a few cases and thoracic surgery did not exist. Whether these methods are sufficient to prevent an increase in the disease cannot be answered yet.—F. Kellerman, M.D., Tubercle, Sept. 1941.

THE MANAGEMENT OF THE EARLY DIABETIC PATIENT

J. C. PASS FEARRINGTON, M. D.

WINSTON-SALEM

The management of the early diabetic is the most important phase of diabetic control. There should be a careful evaluation of mental, physical, and economic factors, as well as a complete history, careful physical examination and laboratory study. Treatment includes directing the diet and teaching the patient to adapt himself to his environment. The family of the diabetic patient should be instructed as to the preparation of the diet and the use of insulin. The patient should realize that he can be a normal individual by accepting his condition and its limitations, and managing it properly.

Diagnosis

The doctor's first problem is the diagnosis of the condition. Joslin has well said that anyone must be considered a diabetic whose urine reduces Benedict's solution until he is proved otherwise. The diagnosis is easily confirmed by the finding of abnormal venous blood sugar levels. A blood sugar over 130 mg. per 100 cc. before breakfast and over 170 mg. per 100 cc. after meals is considered high. It must be remembered that the diet of the patient for two or three days preceding such tests must contain a sufficient amount of carbohydrate to give a fair blood sugar reading. Persons who suspect diabetic conditions often resort to diets high in fat and low in carbohydrate.

Positive urine and blood tests are usually confirmed by the history of weight loss, increased thirst, frequency of urination and increased hunger. A further aid to the doctor is a family history of diabetes, or a history of other endocrine disorders or height and weight abnormalities in the patient or his family. The average duration of life in the family is also relevant.

Age Groups

Diabetics may be divided into three groups according to ages: those under 20, those from 20 to 40, and those over 40. Each group has different problems and requires a different approach.

In patients under 20 there is frequently a hereditary factor. Forty-seven per cent of my patients in this group gave a family history of diabetes. Only 4 per cent were overweight. Severe types of diabetes are frequently met in this group, but improvement is rapid with proper care. Local and general infections are important factors. It is desirable for the patient to maintain his weight at the average level or slightly above, because of the possibility of tuberculosis. The psychological factors of adolescence make this group a most difficult one to handle.

In the group from 20 to 40 years of age hereditary factors were present in 33 per cent of my patients, and 19 per cent were overweight. These patients should maintain approximately the average weight for their sex, age, and height. They should be warned of the dangers of marriage into a family with a diabetic history. Special care is necessary for pregnant diabetic women to guard against toxemia, and the infant should receive particular care following delivery.

In the group over 40 hereditary factors were found in only 24 per cent, but 81 per cent were overweight. Care should be taken to prevent cardiovascular accidents by reducing weight, maintaining low blood cholesterol, and giving particular attention to the cardiovascular system and the extremities.

Treatment

Laboratory tests of the blood and urine should be made for all ages of diabetics. In addition, the following practices are essential:

1. Teaching the patient to make his own qualitative Benedict tests and Gerhardt aceto-acetic acid test.
2. Occasional tests for blood sugar after meals.
3. Periodic tests for blood cholesterol.
4. Occasional twenty-four hour quantitative urine sugar tests.

The last procedure is of especial importance since we are using today slower, longer-acting insulins such as protamine zinc insulin, which acts over a period of twenty-four to thirty-six hours. Single urine and blood sugar tests, although of some

Read before the Section on the Practice of Medicine, Medical Society of the State of North Carolina, Pinehurst, May 20, 1942.

value, are not of the same significance as with regular insulin.

Diabetic patients under 20 years are usually underweight before developing diabetes. In view of this fact and the fact that this is the age period in which tuberculosis is most frequently found, these patients should have a routine tuberculin test, followed by chest x-rays in those with a positive reaction. In the group from 20 to 40 years the weight follows the normal standards.

Overweight is found in 76 per cent of patients over 40 before the onset of diabetes. This excessive fat strains the cardiovascular system, and necessitates special laboratory study and tests for cardiovascular efficiency, such as electrocardiograms, functional heart tests, and functional tests of the extremities.

Pregnancy in the diabetic patient calls for particular care. Hormone studies are valuable. Prolan, serum estrin and urinary pregnandiol tests indicate the necessity for substitution therapy. Dr. White[1] has cut fetal fatalities in diabetic patients from 32 per cent in 1936 to 6 per cent in 1940 by such studies at the New England Deaconess Hospital. Her practice has been to perform cesarean section just before labor is due and to give glucose to the baby for two or three days following birth in order to prevent the hypoglycemic reactions which occasionally occur in children of diabetic mothers. In the care of eleven diabetic mothers I have not employed hormonal studies, but have used estrone and progesterone in large quantities according to clinical indications. Cesarean sections were done by Dr. Richard Spicer and glucose was given to the babies. We have lost no mothers or babies and have had no toxemias of pregnancy.

The handling of a diabetic child has a psychological aspect. Although the child should understand that his food must be simple and carefully calculated and weighed, some responsible adult should see that the food is prepared properly and that the child's environment is normal. With the school child, arrangements must be made for between-meal lunches and observation of the child for insulin shock.

For older diabetic patients the employer should provide time for between-meal lunches. The status of diabetics in industry is yet uncertain, because of the dangers of hypoglycemic reactions in a person handling machinery. A recent article[2] suggests the maintenance of high blood sugars in order to guard against hypoglycemic reactions, and questions the general belief that high blood sugars are damaging. The authors state that their patients in whom the blood sugar level was kept high showed no diabetic symptoms.

The doctor must emphasize to the patient the importance of the following points:

1. Qualitative urine tests for sugar and aceto-acetic acid test.
2. Proper injection of insulin.
3. The prevention and correction of insulin shock resulting from over-activity.
4. Care of the extremities against infection and early treatment of foot infection.
5. Care of acute infections, such as colds and boils.
6. Weighing of foods and preparation of the diet.

I do not believe that people can ever estimate food weights. Therefore I compel all diabetic patients, regardless of age, occupation, or the severity of the disease, to buy scales and use them. Volume measuring of foods is very inaccurate by comparison with weighing. Accuracy is more important than ever with the use of protamine zinc insulin.

The arrangement of the diabetic diet is illustrated in the following typical case reports.

Case 1. A Mild Diabetic Without Symptoms

The patient was a male, 40 years of age. His height was 5 feet, 9 inches; his weight, 190 pounds. A positive reaction for sugar was discovered in an insurance examination. The hereditary factor was absent, and there were no diabetic symptoms. The patient's mother died of a cerebral accident at the age of 49. She was 5 feet 2 inches tall and weighed 170 pounds. His father, height 5 feet 7 inches, weight 220 pounds, died of a coronary occlusion at 54.

The patient's blood sugar before breakfast was 140 mg. per 100 cc.; after breakfast, 190 mg.

Theoretically the patient's weight should have been 162 pounds. With a 5 per cent reduction for cardiovascular safety, his weight should ideally be 154 pounds, or 70

1. White, P., and Hunt, H.: Prediction and Prevention of Pregnancy Accidents in Diabetes, J. A. M. A. 115:2089 (Dec. 14) 1940.

2. Tolstoi, E. and Weber, F. C., Jr.: Protamine Zinc Insulin: A Clinical Study, Arch. Int. Med. 66:670 (Sept.) 1940.

kilograms. (To convert pounds into kilograms, multiply by .45.) The caloric requirement per kilogram for moderate activity is 30 calories, which would mean that this patient required 2100 calories per day. The daily protein requirement is 1 Gm. per kilogram, or 70 Gm. of protein for this patient. One gram of protein is equal to 4 calories, so he should receive 280 calories of protein daily. When this is subtracted from 2100 (the total caloric requirement), 1820 calories are left to be supplied in carbohydrate and fat. The ratio of carbohydrate to fat in these diets is 2:1, 1 Gm. of carbohydrate is equal to 4 calories, and 1 Gm. of fat gives 9 calories. We obtain the carbohydrate-fat factor in the following manner:

2 (Gm. of carbohydrate) × 4	**= 8**
1 (Gm. of fat) × 9	**= 9**
Carbohydrate: fat factor	**17**

Dividing this factor into the number of calories to be supplied in fat and carbohydrate (1820), we obtain a factor divisor of 107 Gm., which is the total amount of fat and one half the amount of carbohydrate the patient should receive. The patient, therefore, was told that he should eat 214 Gm. of carbohydrate, 107 Gm. of fat, and 70 Gm. of protein daily. This was divided among his six daily meals in the following manner:

Breakfast:

Carbohydrate—25 Gm.
Protein—11 Gm. (1/6 the daily ration)
Fat —17 Gm. (1/6 the daily ration)

10 a.m. lunch—10 Gm. carbohydrate (This, plus the 25 Gm. for breakfast, is 1/6 the daily ration.)

Dinner:

Carbohydrate—50 Gm.
Protein—22 Gm. (1/3 the daily ration)
Fat —34 Gm. (1/3 the daily ration)

3:30 p.m. lunch—20 Gm. carbohydrate (This, plus the 50 Gm. for dinner, is 1/3 the daily ration.)

Supper:

Carbohydrate—70 Gm.
Protein—25 Gm.
Fat—45 Gm.

Bedtime lunch:

Carbohydrate—36 Gm.
Protein—10 Gm.
Fat—8 Gm.

These amounts plus the supper allowance equal ½ the total daily ration of each type of food.

The ratio of carbohydrate to fat in this diet (2:1) may be considered high in view of Allen's work[3] showing that tolerance was maintained and improved by undernutrition and low carbohydrate diet. Haist, Campbell and Best[4] also showed that diabetes can be prevented in dogs given anterior-pituitary extract by a low carbohydrate diet, undernutrition, and insulin. I believe, however, that the importance of carbohydrate in preventing early arteriosclerosis outweighs the possible loss of some tolerance.

It has been found that patients with impaired sugar tolerance tolerate foods less well at breakfast, but that tolerance improves as the day progresses, and that the carbohydrate-protein lunch before retiring increases the patient's tolerance for breakfast the next morning.

I have found by sad experience that this group of asymptomatic patients must be treated as real diabetics. Their foods must be weighed, the urine tested, and a slow weight loss encouraged in order to maintain and improve sugar tolerance. Probably insulin will not be needed, but the patient will need guidance and cardiovascular studies. In brief, our aims in dealing with this group are the following:

1. To adjust the patient's weight to the desired level to improve or maintain his insulin reserve.

2. To defer, minimize and treat early cardiovascular accidents to which he is heir.

We must minimize diabetes and arteriosclerotic factors by giving high carbohydrate to fat ratios, reducing weight, limiting the cholesterol content of the diet, reducing nervous and physical tension, and guarding against damage to extremities. This patient is approaching the age-range in which both of his parents succumbed to cardiovascular disease.

Case 2. The Patient With Early Diabetic Symptoms

This case is very much like the first except that the patient had the diabetic symptoms of weight loss and increased thirst and hunger. A positive urine sugar was discovered by a local doctor. The hereditary factor was absent. The patient's mother and father were both living at 65. The mother had hypertension, and was 5 feet 3 inches tall and weighed 155 pounds. The father,

3. Allen, Frederick, M.: Studies Concerning Diabetes, J. A. M. A. 63:939 (Sept. 12) 1914.
4. Haist, R. E., Campbell, J., and Best, C. H.: Prevention of Diabetes, New England J. Med. 223:607 (Oct. 17) 1940.

who had some evidence of coronary disease, weighed 185 pounds and was 5 feet 8 inches tall.

The patient's blood sugar was found to be 170 mg. per 100 cc. before breakfast and 220 mg. after breakfast.

The patient's ideal weight was estimated to be 154 pounds, the same as that of the first patient. The diet, therefore, is identical with that outlined in case 1. In this case, however, substitution therapy is necessary. Protamine zinc insulin is given at breakfast, and the patient's urine is checked before and after meals. After the urine is found to be negative throughout the day (unless complications develop) the blood sugars are checked before breakfast, at 10 a.m., and at 3 p.m. The patient's activity and other factors which might produce insulin shock are regulated.

This type of diet and protamine zinc insulin correct 55 per cent of all my diabetic cases, but the disease must be reasonably mild and the patient's habits regular.

Case 3. A Juvenile Diabetic

The patient was a 15 year old male, height 5 feet 11 inches, weight 135 pounds. A positive urine reaction for sugar was discovered by a doctor who examined the patient because of his tired feeling and increased thirst. The patient's father and maternal uncle had diabetes. His mother was 45 years old, and in good health. Her height was 5 feet 2 inches, her weight 135 pounds. The father was 48, and was found to be diabetic at 41. He was 5 feet 11 inches tall and weighed 175 pounds.

The patient had lost 17 pounds in two months and complained of increased thirst and urination, and muscle cramps at night. His blood sugar was 190 mg. per 100 cc. before breakfast and 250 mg. after breakfast.

Theoretically the patient's correct weight was 154 pounds, or 70 kilograms. A boy's caloric requirement is 40 calories per kilogram—2800 calories for this patient. His protein requirement is 1½ Gm. per kilogram, which would be 105 Gm. or 420 calories of protein per day. The carbohydrate-fat ratio is again 2:1, and 2380 calories are left to be supplied in carbohydrates and fats. This number is divided by the carbohydrate-fat factor of 17 (obtained as in case 1), giving a factor divisor of 140 Gm. Therefore the patient's daily diet must contain 280 Gm. of carbohydrate (140 × 2), 140 Gm. of fat (140 × 1), and 105 Gm. of protein. One third of the daily ration is served at each meal, with 30 per cent of the dinner allowance being moved to 10 a.m. Breakfast and dinner must be served within four hours.

Substitution therapy was also necessary for this patient, and he was begun on regular insulin, 40 units at breakfast and 20 at supper. This dosage was regulated according to the urinary findings, blood sugars and symptoms.

The adolescent type of case should respond well to treatment, and tolerance will be improved by maintaining low blood sugars. Adolescent diabetics are difficult to handle, for they assume no responsibility. It is not always the best procedure to remove the patient from all athletic activities. This boy, who is in school in the morning at a sedentary occupation, will have most of his food at this period, and his blood sugar will be controlled by regular insulin, which will be practically consumed during this period. In the afternoon, when he is participating in athletics, he will burn up much of his carbohydrate reserve, yet he will run less risk of reaction from regular insulin (which is usually used in four to six hours) than from crystalline or protamine zinc insulin, which is always a menace under severe physical strain.

Type of Insulin

There are some diabetics whose dose of protamine zinc insulin will control blood sugar during the day, but who will become hypoglycemic in the early morning, even if 50 per cent of the evening allowance of carbohydrate and protein is given at bedtime. This group is known as Woodyatt's "brittle" type of case. For these patients we must mix protamine zinc insulin with quicker and more limited types of insulin. Regular insulin is effective over a period of five to seven hours when used with protamine zinc insulin, and the effect of crystalline insulin is felt for twelve to fourteen hours when it is used with protamine zinc insulin.

The type of insulin employed with protamine zinc insulin depends upon the manner in which the food intake is divided. If the patient prefers a large breakfast and dinner with a small supper, a large morning lunch, and a small evening lunch, give regular insulin plus protamine zinc insulin at breakfast.

If he eats a moderate breakfast with a large dinner and supper and lunches between meals, give crystalline insulin plus protamine zinc insulin at breakfast. In the first group the following rules should be applied:

1. Use three times as much regular insulin as protamine zinc insulin (30 units of regular and 10 units of protamine zinc insulin).
2. Increase protamine zinc insulin until the urine tests at 10 p.m. and before breakfast show a green to yellow (qualitative Benedict's) reaction.
3. Increase regular insulin until the tests before and after meals are negative.
4. Increase protamine zinc insulin until urine tests before breakfast and at 10 p.m. are negative.

Abstract of Discussion

Dr. O. Norris Smith (Greensboro): My first reaction to Dr. Fearrington's paper was that it makes the treatment of diabetes sound more difficult than it is.

I thoroughly agree with Dr. Fearrington that every overweight diabetic patient should have his weight reduced by dietary control. One hospital in Philadelphia has two diabetic clinics in two different medical services. The man in charge of one clinic follows the hypothesis of Allen that weight should be reduced. In the other clinic the chief for some reason prefers to allow the patient to eat excessively and maintain weight. Sixteen per cent of his patients require insulin in large doses.

There are one or two points that I would like to bring out. First is the importance of an optimistic approach to the diabetic patient. In Greensboro a year ago two diabetics committed suicide within a period of three months. I am glad that neither of them was my patient.

In the diabetic woman insulin should be omitted during early pregnancy, and should then be increased from time to time. Very shortly after delivery, the dose can be returned to almost the initial level.

I should like to emphasize the procedure outlined by Haist, Campbell and Best in the **New England Journal of Medicine** last fall. Their work was experimental but it was extremely suggestive. They found that a low calorie diet and the early use of insulin in controlling diabetes materially increases the amount of carbohydrate tolerance that the patient regains. We all know that every patient who goes into diabetic coma loses some carbohydrate tolerance and requires larger doses of insulin subsequently. There are several practical considerations on which I disagree with these authors, however. One is the lack of urine check following protamine zinc insulin and the disregard of the blood sugar unless it is excessive, in which case they increase the dose of protamine zinc insulin and recommend a bedtime meal to guard against shock in the morning.

I believe that actual weighing of food is necessary in a very small proportion of diabetic patients. Doing away with this procedure is certainly part of the optimistic approach. I have used with complete satisfaction a group of stock diets suggested by the North Carolina Dietitians Association. They are using them in a great many hospitals in the state. These diets are made up for any number of calories you want. I certainly do not feel that it is necessary for a doctor to sit down and laboriously construct a diet for every diabetic patient, when a stock diet properly used will suffice in certainly 95 per cent of the cases.

Dr. L. A. Crowell, Jr. (Lincolnton): With some things that Dr. Fearrington said I agree heartily, and with some I disagree. Some of the things I am going to say will probably be considered unscientific, but I have a series of nearly 300 living diabetic patients who, I believe, are doing as well as the average. The first thing I want to discuss is the diet. It is well to know how to sit down and figure out theoretically the proper diet for each individual patient, but there are many variations that we cannot figure. I have a series of diets ranging from about 1000 calories up to 2500, one of which I give the patient when he first comes into the hospital. I always start the diet and insulin on the same day. It used to be the practice to decrease the diet gradually until the patient's urine was sugar free, then build up the insulin dose and the diet at the same time. I think that this practice is a waste of time. We put the patient on a diet roughly estimated to meet his requirements. No attempt is made to get the exact scientific amount, and the diet is governed by the patient's appetite or weight curve and his sense of well being. The insulin is then regulated to suit the diet.

One thing I do insist on is that every diabetic have a diabetic scale. Every one of the 290 patients I have treated have bought diabetic scales, and I suppose I have as high a percentage of indigent patients as anybody.

Dr. W. Raney Stanford (Durham): It has been my practice to regulate the diet and then fit the insulin dosage to it. In very high carbohydrate diets the patient is a little more likely to get insulin reactions. I use a moderate carbohydrate diet, usually 150 or 160 Gm. a day. I think that is about what Joslin uses.

With protamine zinc insulin we gradually increase the dose until the morning blood sugar is somewhere between 120 and 140 mg. per 100 cc. If it is lower, the patient might get a reaction.

Dr. Fearrington: As to weighing food, I have found out that the people who do not weigh food get in trouble, and that those who do weigh it are much better regulated.

Dr. Crowell's idea of supplying diabetic patients with a diet already worked out may be all right, but I think that every doctor ought to know the scientific basis for such a diet.

The juvenile diabetic now lives to hand the disease down to more juveniles. I have 53 juvenile diabetic patients who will transmit the disease to well over 1000 children. The problem of diabetes, then, is bound to increase.

The Peritoneoscope. — We have no desire to possess a peritoneoscope. We have seen it used elsewhere. It is brutal and futile. The shock of inflating the peritoneal cavity with air and the danger of perforating the gut are too great and the keyhole view is unsatisfactory even for the most ardent Peeping Tom.—William J. Carrington: Differential Diagnosis of Acute Lower Abdominal Lesions in the Female, J. M. Soc. New Jersey 38: 504 (Sept.) 1941.

THE TREATMENT OF BURNS WITH A SPRAY OF SULFADIAZINE

Report of a Case

W. EUGENE KEITER, M. D.

KINSTON

C. T., a colored male, aged 18 months, fell backward into a tub of scalding water on January 14, 1942. He was brought to the Memorial General Hospital at Kinston within an hour of the accident. The skin was blistered and sloughing over an area extending from the neck to the popliteal regions posteriorly, over the lateral aspects of the thighs and abdomen, over the anterior and upper inner aspects of the thighs, the genitals, and lower abdomen. The child was crying with pain but was not in shock. He was trembling and shivering with cold and excitement.

The loose, pigmented skin was removed by grasping it with sterile gauze. It came off in sheets, leaving the deep layers pink. As fast as it was removed an assistant sprayed the area with an aqueous solution of sulfadiazine 3 per cent and triethanolamine 8 per cent[1]. The child was put under a light cradle and the spray was applied almost continuously during the first two or three hours, and then at intervals of one hour. Opiates were not given, since the spray seemed to ease his pain. Fifteen hundred units of antitetanic serum were given.

An intravenous cannula was put in the left saphenous vein anterior to the internal malleolus and 5 per cent dextrose in Ringer's solution was given as a continuous intravenous drip. Two hundred cubic centimeters of citrated blood were given through this cannula about three hours after the accident and again the following morning, after which the intravenous drip was discontinued.

The first outstanding feature in the child's

1. Pickrell, K. L.: New Treatment of Burns (Spraying Solution of Sulfadiazine, Sulfanilamide Derivative); Preliminary Report, Bull. Johns Hopkins Hosp. 69:217 (August) 1941.

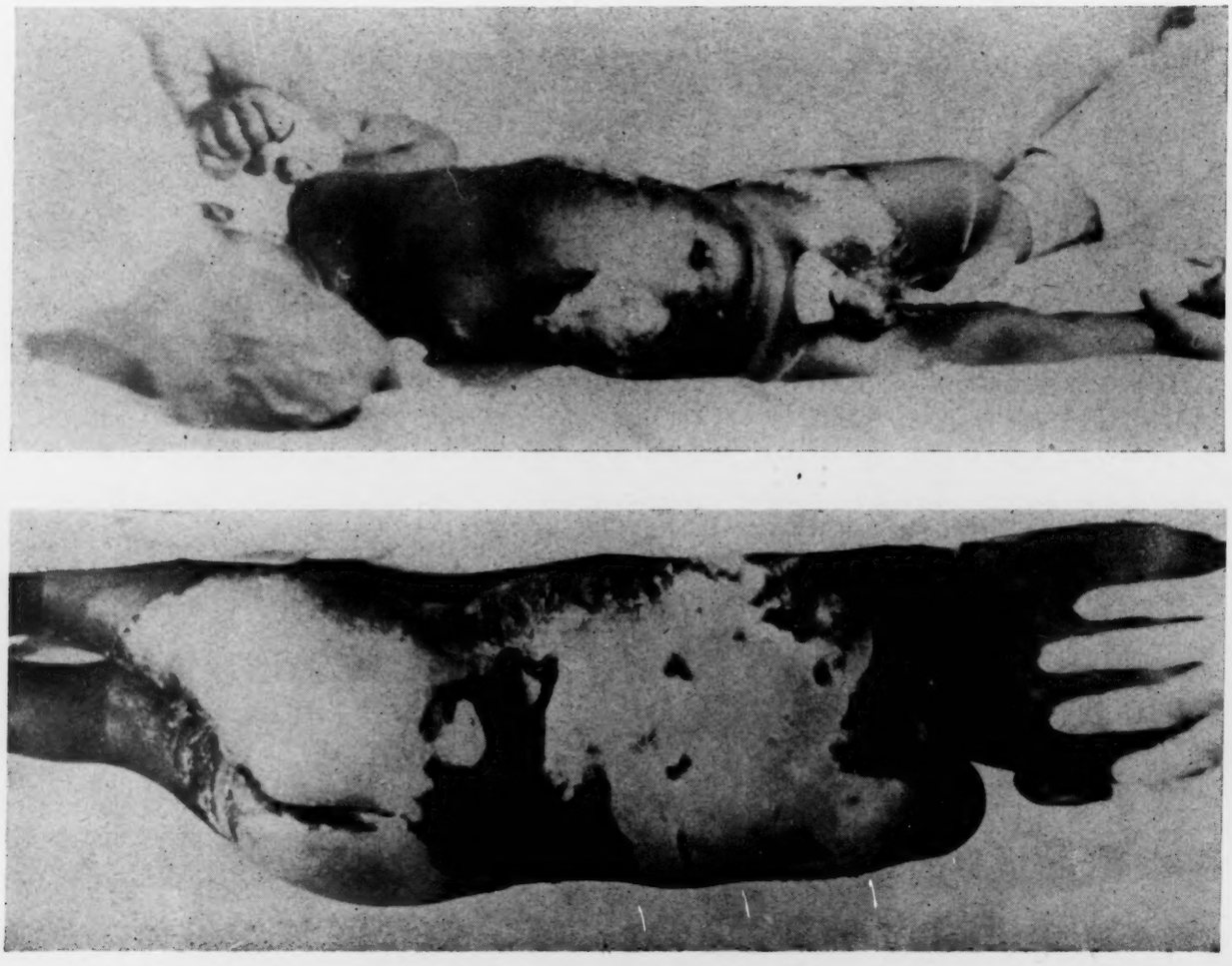


Fig. 1. C. T. on January 17, 1942.

progress was a surprising absence of toxic symptoms during the first three days. These were of course combatted to a great extent by the intravenous drip and the two transfusions given during the first twenty-four hours. In our previous experience with burns of comparable extent, however, we have always encountered severe toxemia despite fluids and transfusions. Therefore we do not believe that these general measures were altogether responsible for the absence of toxic symptoms.

The second outstanding feature was the absence of infection in the burned area. The spray caused a beautiful transparent eschar, not unlike pliofilm or cellophane in appearance, to form. Islands of epithelial regeneration showing black pigment appeared within the first four or five days. These were plainly visible through the eschar. The child voided on his thighs, wetting the bed frequently. He developed a diarrhea on January 20 and frequently soiled the bed and the burned area with feces, yet no serious infection ever occurred in the burned area about the genital region. We attributed this to the continuous cleansing of the region with peroxide and spraying with the sulfadiazine mixture.

The third outstanding feature was the rapidity of healing and the short period of hospitalization. As fast as the eschar sloughed White's "A and D Ointment" was applied. This apparently hastened epitheli-

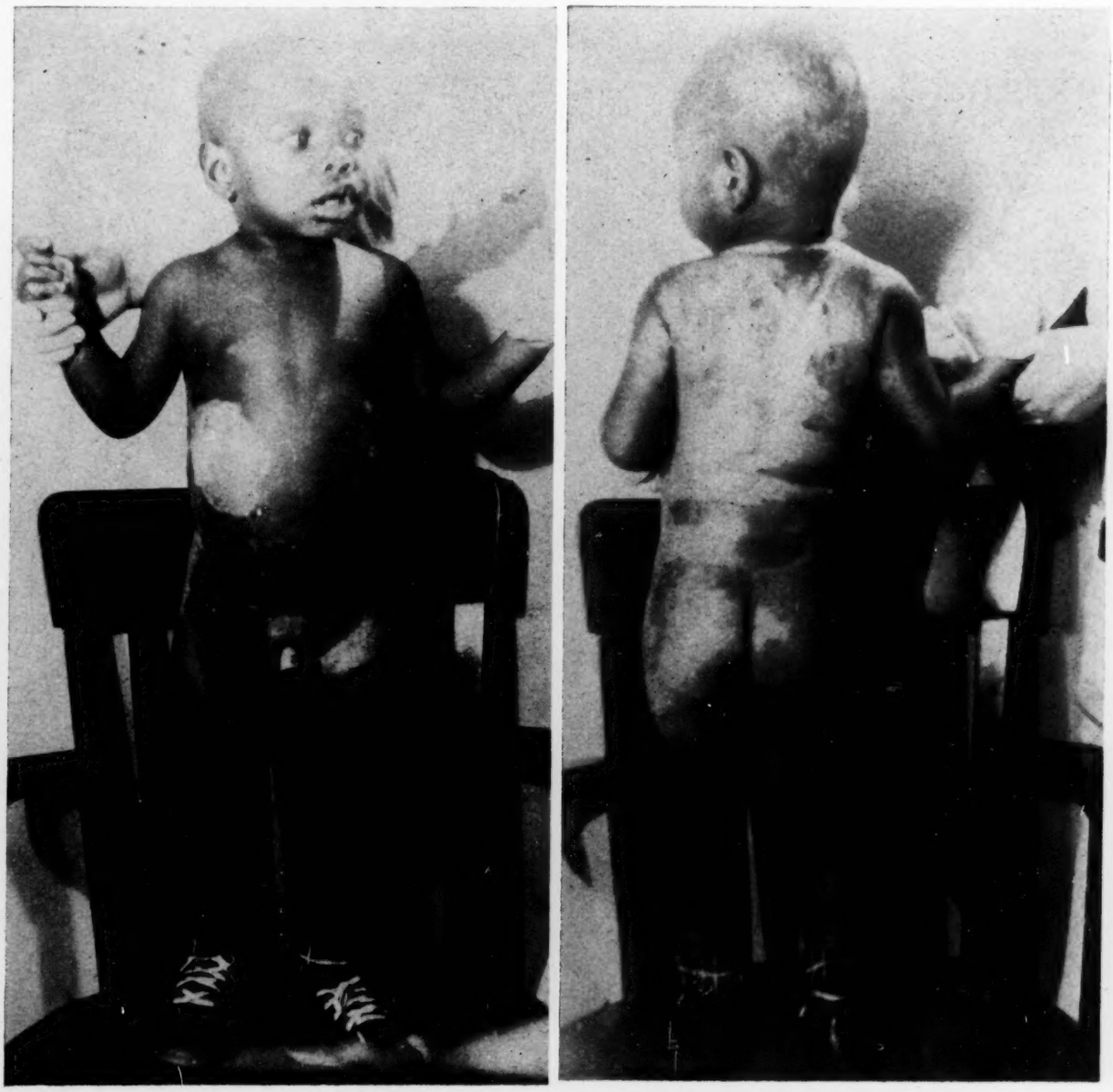


Fig. 2. C. T. on February 5, 1942.

zation. The eschar began sloughing on the fourth or fifth day. On February 4 the child was allowed to be up about the hospital, wearing clothes. He was discharged fully dressed on February 7.

The chief advantage of this treatment seems to be in the early stages: in allaying pain, preventing toxemia, and combatting infection.

I wish to thank Miss Mildred Ringle, Medical Technologist, Memorial General Hospital, for photographing the child. I also wish to thank Dr. Fleming Fuller for helping with the debridement and first application of the treatment.

MECKEL'S DIVERTICULUM AND ITS COMPLICATIONS

JAMES F. MARSHALL, M. D.

WINSTON-SALEM

During the past ten years reports on Meckel's diverticulum and its complications have become increasingly frequent in the literature, and correct preoperative diagnoses have become much less rare.

Lavater reported observations on a diverticulum of the terminal ileum in 1671, and Ruysch, in 1707 and Morgagni in 1769 discussed this condition. However, Meckel (1781-1833) is given credit for the association of the diverticulum with the omphalomesenteric or vitelline duct.

Normally this structure disappears during embryological development, but in about 1.5 per cent of individuals it persists, varying from an outpouching on the antimesenteric border of the lower ileum to a continuous tract with an external opening at the umbilicus. Frequently the diverticulum is attached to the peritoneal side of the umbilicus by a fibrous band, and at times the structure is attached flush to the under side, with no opening to the exterior. It contains all the coats of the ileum and, in addition, gastric mucosa and pancreatic tissue at times. Gastric mucosa is present in 13 per cent of the cases, according to Curd[1], who studied non-pathologic cases at autopsy. However, many writers have recorded much higher figures; Holmes and Koplik found gastric mucosa in 67 per cent of pathological cases. This structure is usually 50 to 90 cm. from the ileocecal valve, although the distance varies from a few centimeters to 192 cm. One case has been reported in which the duct arose from the appendix itself[2].

This anomaly occurs much more frequently in males. The ratio of occurrence in males and females is about 3:1. No plausible reason has so far been advanced for this disproportion.

The majority of writers find that complications are most apt to be discovered in children under 15 years of age. Cobb[3] reported 74 per cent of his cases of Meckel's diverticulum with peptic ulcer to be in patients under this age, and most of these to be under 5.

There are four types of pathologic lesions found most frequently at operation: (1) Simple peptic ulcer or ruptured peptic ulcer; (2) obstruction; (3) inflammation; (4) rarely, some type of tumor.

The most common complication reported is that of peptic ulcer. Miller and Wallace[4] in a group of 201 cases abstracted from the literature found that 46.2 per cent had peptic ulcer. Prior to rupture the outstanding symptom is blood in the stool. Cobb[3] reported that 72 per cent of the 100 cases analyzed by him presented this symptom at one time or another. The blood is usually dark red and unmixed with stool, and often contains clots. Prior to, or at the time of passage, there is sometimes colicky pain in the lower abdomen or about the umbilicus. These patients usually have two or more hemorrhages before being operated upon, and nearly always show secondary anemia.

Diverticulitis *per se* is apparently less frequently a complication than was formerly believed. Miller and Wallace[4] found only 4.47 per cent of their group of cases to be inflammatory. On the other hand, Womack and Siegert[5] reported that 21 per cent of their 19 cases were inflammatory. These figures exclude, of course, that large group of cases of ulcer and its complications. The symptoms of diverticulitis are those of an inflammatory reaction in the region of the umbilicus or a little to the right or left. Pain usually begins about the umbilicus or to the right of it, less frequently to the left.

Read before the Forsyth County Medical Society, Winston-Salem, October 14, 1941.

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3. Cobb, Donnell B.: Meckel's Diverticulum With Peptic Ulcer, Ann. Surg. 103:747-764 (May) 1936.

4. Miller, Richard H. and Wallace, Richard H.: Meckel's Diverticulum in Acute Abdominal Emergencies, Ann. Surg. 98:713-721 (Oct.) 1933.

5. Womack, Nathan A. and Siegert, R. B.: Surgical Aspects of Lesions of Meckel's Diverticulum, Ann. Surg. 108: 221-236 (August) 1938.

It is often colicky in nature and is followed by nausea and vomiting. There is leukocytosis and later fever. Locally, there are muscle spasm and tenderness, with rebound tenderness appearing later. Following rupture the signs are those of peritoneal soiling. In some cases they tend to localize and in some they do not. Mention should be made of the fact that diverticulitis is sometimes complicated by some form of obstruction.

It should also be noted that in children peptic ulcer is the most common complication of Meckel's diverticulum, whereas in adults most of the reports concern some type of obstruction or inflammatory reaction.

It is readily understood how intestinal obstruction can occur as a complication when one visualizes the duct attached to the umbilicus itself or to some other area in the abdominal cavity by a band. It is easy to see how a loop may slip beneath the band and become obstructed, or a volvulus may occur, with the fixed distal attachment acting as a turning point. Intussusception also occurs and is more frequent in children in those cases without a fixed attachment. Twenty-six per cent of Womack and Siegert's cases[5] had some type of obstruction. Of the cases analyzed by Miller and Wallace[4], 44 per cent presented some type of obstruction.

The finding of a tumor in patients with Meckel's diverticulum has been rare. They are usually "carcinoid" tumors, and occasionally lipomas and fibromas occur. Nygaard and Walters[6] found reports of 6 cases of adenocarcinoma and 12 cases of sarcoma in the literature and added 2 more cases of sarcoma. These cases showed no evidence of metastasis, although there had been recurrences in 2 cases, both five years after removal. Some of them presented no symptoms, some had abdominal "distress" or pain, and in some a palpable tumor was present.

The diagnosis of Meckel's diverticulum seems to be quite difficult. However, in children with blood in the stools, abdominal cramps, and secondary anemia with no evidence of blood dyscrasia, the diagnosis should be made in a high percentage of cases. In general one must have a high index of suspicion for this lesion to diagnose it preoperatively. In inflammatory lesions with pain and tenderness near the umbilicus (most often to the right), which are not typical of acute appendicitis, one should certainly consider this condition. It is well to note that the usual diagnosis is that of acute or "chronic" appendicitis.

In patients presenting signs and symptoms of intestinal obstruction, particularly in those under 40 and with no obvious etiological factor, one should suspect this condition.

An uncomplicated and unpalpable tumor will probably not be diagnosed except by x-ray.

Pfahler in 1934[7] published an x-ray picture of a Meckel's diverticulum filled with barium. He made the diagnosis and verified it at operation. Since then other reports have appeared in which this condition was diagnosed roentgenologically[8]. Pfahler says that these cases show characteristics belonging to any intestinal diverticulum. "The diverticulum cannot be filled with barium mixture if it is already filled with some other material. It is apt to show definite peristalsis. It is smooth in outline and it retains its position at a fixed point. Examinations should be careful and repeated. Observations should be long and made in every position." Following suspected rupture a flat plate or lateral plate will sometimes show pneumoperitoneum.

As is to be suspected the mortality of operation varies. Hudson reported an operative mortality of 22 per cent; Cobb (with peptic ulcer) of 27 per cent[3], and Everhart of 57 per cent[9]. The mortality varies with the complication present, the length of delay before operation, and the skill of the surgeon. In general the earlier the condition is attacked, the lower will be the mortality.

In the 12 cases operated on at the City Memorial Hospital from January, 1936, to October, 1941, there were no deaths. The operations were performed by several different men. The series contained no case of perforation. The type of operation employed was excision and inversion, and in one case, resection. Simple excision carries the lowest mortality, but a small amount of

6. Nygaard, K. K. and Walters, Waltman: Malignant Tumors of Meckel's Diverticulum, Arch. Surg. 35:1159-1172 (Dec.) 1937.

7. Pfahler, George E.: The Roentgenological Diagnosis of Meckel's Diverticulum, Surg., Gynec., and Obst. 59:929-934 (Dec.) 1934.

8. Ehrenpreis, Bernard: The Roentgen Diagnosis of Meckel's Diverticulum, Am. J. Roentgenol. 42:280-284 (August) 1939.

9. Everhart, Merrill W.: The Complications of Meckel's Diverticulum in Infancy and Childhood, J. Pediat. 17: 488-489 (Oct.) 1940.

gastric mucosa may be left behind in some instances. It seems to me, however, that in the acute cases the simplest operation is the procedure of choice.

The youngest patient in this group was 7 months old and the oldest 50. Only 3 were under 15 years of age, and the average age was 22.8 years. There were 9 males and 3 females—a ratio of 3:1.

Symptoms included colicky abdominal pain, nausea and vomiting in the cases of obstruction, bleeding in one case, and anorexia in one elective case in which operation was for retroversion of the uterus and "chronic appendicitis".

Each case with definite complicating pathology showed a leukocyte count above 13,000, except for the case with bleeding and one case in which the count was not recorded. The highest count was in one of the cases of volvulus and was 24,000.

The correct diagnosis was made in only one case, a baby 1 year old with bleeding from the bowel. In one other case a correct diagnosis of intestinal obstruction was made, but the etiological factor was not suggested. In another case of appendiceal abscess with secondary involvement of a Meckel's diverticulum the abscess was diagnosed correctly, but there was no reason to suspect the presence of the diverticulum. It is of interest that acute or "chronic" appendicitis entered into the diagnosis of every case except 2. In only 2 cases was the appendix really involved, and in these cases the Meckel's diverticulum was an incidental finding. Four of the cases gave a history of previous symptoms.

Of the 12 cases 7 showed definite pathologic lesions of the diverticulum. There were 4 cases of intestinal obstruction. Volvulus was found in 2 cases with the distal attachment to the umbilicus. In one case the distal attachment was to another loop of ileum and in the other it was to the mesentery, through which a loop of ileum had slipped, producing obstruction. There were 2 cases showing acute inflammatory reaction. One of these was primary and the other was secondary to an appendiceal abscess. Unfortunately, in the case of melena in the year old baby, the pathologist failed to demonstrate an ulcer.

All except 2 cases were treated by excision

Case	Sex	Age	Preoperative Diagnosis	W. B. C. Count	First Symptom	Location of Pain	Previous Attacks	Operative Procedure	Hospital Days	Wound Infection	Cm. from Valve	Attachment	Postoperative Diagnosis
1.	M	17	Acute appendicitis	21,600	Colicky pain Vomiting	Umbilicus	None	Excision and Inversion	11	No	92	Umbilicus	Volvulus of Meckel's diverticulum
2.	M	50	Chronic cholecystitis; chronic appendicitis	8,400	Colicky pain	Umbilicus	Indefinite	Excision and Inversion	12	No		Not stated	Meckel's diverticulum
3.	M	25	Acute appendicitis	16,900	Soreness	Epigastrium	None	Excision Inversion, Drain	15	No	92	Not stated	Gangrenous diverticulitis
4.	M	31	Acute appendicitis	None	Epigastric pain	RLQ	Not stated	Excision, Inversion, Drain	16	Yes		Abscess	Appendiceal abscess Subacute diverticulitis
5.	F	23	Chronic appendicitis	8,100	Anorexia	Lower abdomen Back	?	Resection and Anastomosis	16	No		Not stated	Meckel's diverticulum Retroversion of uterus
6.	F	25	Recurrent appendicitis	8,100	Vomiting, Pain	RLQ	Yes	Excision and Inversion	12	No	20	Not stated	Meckel's diverticulum
7.	M	43	Acute appendicitis	13,600	Generalized pain	RLQ	None	Excision and Inversion	11	No		Ileum	Intestinal obstruction Meckel's diverticulum
8.	M	25	Acute appendicitis	24,900	Abdominal pain	RLQ	Indefinite	Excision and Inversion	13	No	60	Umbilicus	Volvulus of Meckel's diverticulum
9.	M	1	Ulcer	8,400	Bleeding		Severe	Excision and Inversion	10	No	60	None	Ulcer of Meckel's diverticulum
10.	F	9	Interval appendectomy following rupture 3 months previously	8,900		RLQ	None	Excision and Inversion	11	No		Ileum	Meckel's diverticulum
11.	M	35	Recurrent appendicitis	13,800	Abdominal pain	Abdomen	None	Excision and Inversion	11	No	20	None	Meckel's diverticulum
12.	M	7 mo.	Intestinal obstruction	9,100	Vomiting for 4 days		None	Excision of Band	9	No		Mesentery	Intestinal obstruction Meckel's diverticulum

and inversion of the stump. The diverticulum was resected in one elective case, and a 7 months old baby who was quite ill with intestinal obstruction due to the Meckel's attachment obstructing a loop of ileum, was treated by excision and inversion of the band and the diverticulum was left *in situ.*

The distance of the anomaly from the ileocecal valve was stated in only 6 cases. In 2 the distance was about 90 cm., in 2 it was 60 cm., and in 2, 20 cm.

Drainage was employed in 2 cases, one of them being the appendiceal abscess. This was the only case in which there was a wound infection. Average hospitalization was twelve days, the longest stay being sixteen days and the shortest nine days.

Conclusion

1. Meckel's diverticulum should be recognized oftener, particularly in those cases with ulcer and hemorrhage, and in those young people with intestinal obstruction for which there is no known etiological factor.

2. The high mortality can be reduced by early operation in the acute cases.

3. In young individuals ulcer is the most frequent complication; in adults obstruction and inflammatory lesions are most common.

MOLDED PLASTER SPLINTS IN THE TREATMENT OF FRACTURES OF THE HAND

GEORGE L. CARRINGTON, M. D.

BURLINGTON

Fractures of the hand require, of course, a variety of treatments and splints, depending upon the location and character of the injury. Fractures of the metacarpal bones not many years ago were put up on such articles as baseballs and roller bandages. More recently light metal splints made by various surgical supply houses and usually listed as "metacarpal splints" have enjoyed a considerable and deserved vogue. Made of light metals, they have been comfortable and, for most fractures, efficient.

There have been two main objections to these commercial splints in our outpatient department. The first has been the failure of the itinerant patient to return the splint. The second objection has been the failure of the splint to fit perfectly the patient's hand.

Both of these objections have been obviated here by the use of molded plaster

From the Alamance General Hospital. Submitted for publication January 24, 1942.

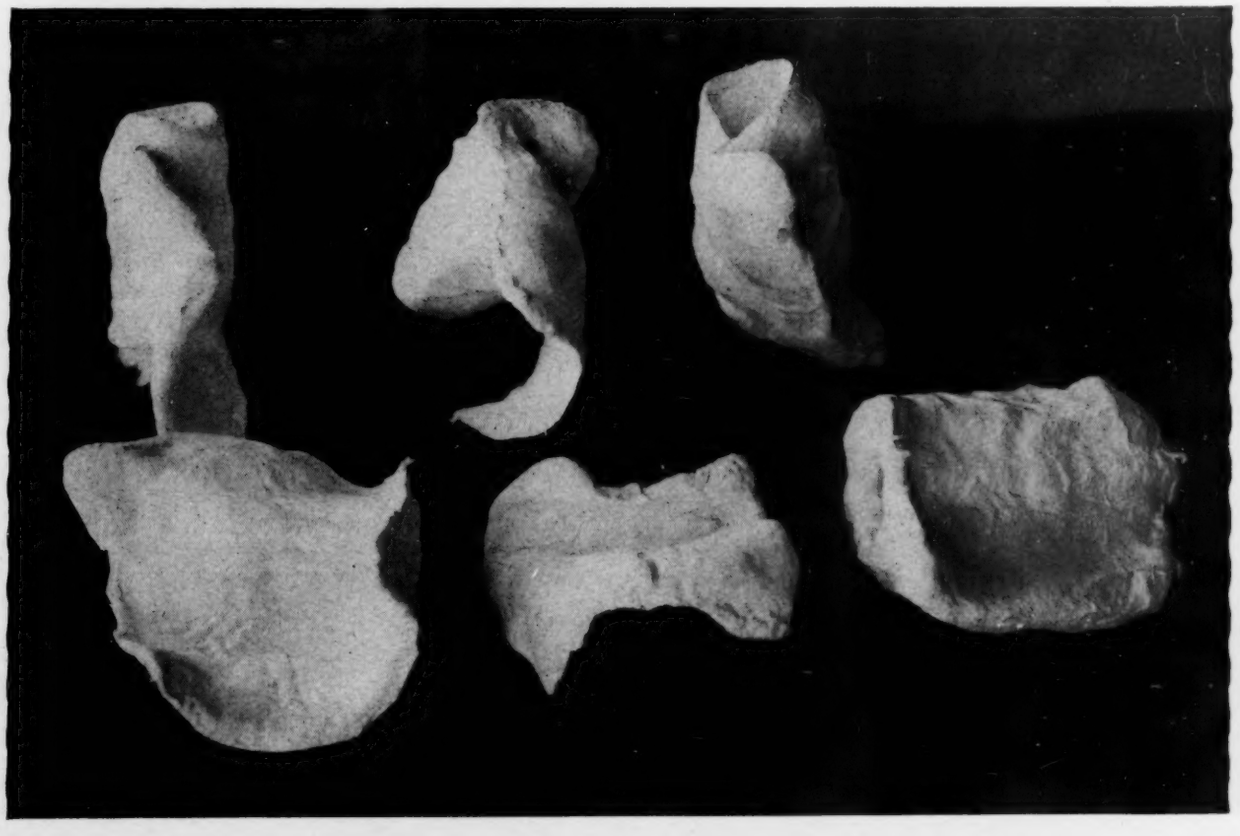

Fig. 1. Molded plaster splints used in treating fractures of the hand.

splints. A single roll of plaster of paris in a few minutes can be molded into a better splint than can be obtained from manufacturers for several dollars. It is an individual splint that is the exact shape desired for the palm of the patient's hand. If the patient fails to return it, there is no great loss.

The illustrations show two types of molded plaster splints. The first were made by wrapping the plaster around a roller bandage and then molding it to the patient's hand. Later the roller bandage was withdrawn. This type has been discontinued in favor of simple flat plaster molded into the patient's hand. The patient's fingers and the plaster are grasped by the doctor's hand, and the plaster is molded over the doctor's knuckles pressed against it into the patient's palm.

This method of making metacarpal splints is probably used in numerous places, although I have not seen it used or written about. The general use of more expensive and less efficient metal splints is the reason for writing this note.

THE TREATMENT OF CHRONIC IDIOPATHIC ULCERATIVE COLITIS WITH SULFAGUANIDINE

DAVID CAYER, M. D.*
and
JULIAN M. RUFFIN, M. D.†
DURHAM

Derivatives of sulfanilamide have been used in the treatment of chronic idiopathic ulcerative colitis for several years[1]. From the Mayo Clinic encouraging results have been reported[1a] with the use of neoprontosil. Other writers, however, have found this drug to be of little value, and our experience with it at Duke Hospital has been discouraging. Sulfapyridine[1b], likewise, has been of doubtful value in the treatment of such patients. It has been the feeling of some observers[1b] that certain sulfonamide derivatives are efficacious in the treatment of ulcerative colitis, but that toxic manifestations often make it necessary to discontinue therapy before the best clinical results can be attained or properly evaluated.

Recently, sulfanilylguanidine (now called sulfaguanidine), a water soluble sulfonamide derivative which is poorly absorbed from the gastrointestinal tract and has a high anti-bacterial activity, has been described[2]. These properties have suggested the possible effectiveness of sulfaguanidine in the therapy of infections localized in the intestines, since high concentrations can be attained in the gastrointestinal tract, while the blood and tissue concentrations remain low. Bornstein and Strauss[3] studied the action of this drug on different Salmonella groups and noted a marked bacteriostatic effect on Escherichia coli, Eberthella typhi, and Shigella, but concluded that its use for types other than the susceptible ones was perhaps contraindicated since the pathogens may flourish while the non-pathogenic organisms are suppressed.

Material

This report is based upon a study of 17 patients having chronic idiopathic ulcerative colitis who were treated with sulfaguanidine. There were 6 males and 11 females ranging in age from 6 to 55 years. The duration of the disease varied from six weeks to nine years. All of these patients were hospitalized, and eight of them had been treated at Duke Hospital on previous occasions. The diagnosis was based upon the characteristic proctoscopic picture, revealing a diffusely inflamed mucosa with pin-point ulcers, or a granular mucosa covered with a bloody exudate. In every case care was taken to exclude such diseases as amebic dysentery, typhoid, bacillary dysentery, carcinoma of the colon, intestinal tuberculosis, and polyposis by stool examination and culture, agglutinations, barium enemas and proctoscopic study. All the cases were considered clinically active at the time of admission.

Submitted for publication January 13, 1941.

* Assistant in Medicine, Duke University School of Medicine.
† Associate Professor of Medicine, Duke University School of Medicine.

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Treatment

The patients were fed a low residue diet supplemented by accessory vitamins, antispasmodics, and general supportive therapy, including transfusions when indicated. Four patients received sulfaguanidine by mouth, one by retention enema alone, and twelve both orally and by retention enema. The oral dose was 2 Gm. three times a day. The retention enemas, containing 5 Gm. of the drug, were given twice daily. Urinalysis, hemoglobin and white cell determinations, and blood sulfaguanidine levels were done every third day. Patients were examined by proctoscope twice weekly. The average duration of treatment with sulfaguanidine was fourteen days.

Results of Treatment

The results of treatment are shown in the accompanying table. Four patients showed a marked improvement following their course of treatment. Three of these were seen several months later and were symptom free. The proctoscopic picture was either normal or showed a granular mucosa without pus or blood. The fourth patient died two months after discharge from the hospital; the cause of death is unknown. In 10 patients some improvement was observed after treatment; however, five of these later had relapses, two of which were fulminating and terminated fatally after ileostomy. In 3 patients no appreciable change was observed at the time of discharge from the hospital, but two of these showed marked improvement six weeks later.

RESULTS OF TREATMENT

	No. of Patients
Marked Improvement	4
Some Improvement	10
(5 relapsed later)	
No Improvement	3
(2 improved later)	
Total	17

Toxicity

Two patients had a generalized rash during the course of treatment, but with these exceptions no toxic symptoms were observed. The urine showed no changes during the period of treatment and the blood picture remained unaffected. The highest blood concentration of sulfaguanidine was 3 mg. per 100 cc., the average being 1 mg. even when the patient was receiving 16 Gm. a day.

Stool Cultures

Repeated cultures of the stool were taken by proctoscopic examination in every case. Before treatment the organisms usually found were hemolytic E. coli, hemolytic streptococci and non-hemolytic E. coli. Bargen's bacillus and an anaerobic mixture were found in only a few cases. After treatment the hemolytic organisms were greatly reduced or absent in every case. The drug had little effect upon the colon bacillus. No correlation could be observed between the disappearance of the hemolytic organisms from the stool and the clinical condition of the patient, nor was there any relationship between the apparent effectiveness of the drug and the duration of the disease.

Discussion

In an effort to evaluate the effectiveness of sulfaguanidine in the treatment of chronic idiopathic ulcerative colitis, 17 patients have been studied. All were clinically active at the beginning of the treatment and all were hospitalized. Although marked improvement was observed in 4 patients and some improvement in ten at the end of the period of treatment, this effect cannot be attributed to the drug alone, as such factors as diet, rest, vitamin therapy and psychotherapy all play a more or less important part in the treatment of this disease.

When the results obtained in this series of patients were compared with those in a group of patients receiving non-specific therapy only, no appreciable differences were observed, nor did the drug prove materially more efficacious than other sulfonamide derivatives which had been used previously in the treatment of ulcerative colitis.

Conclusion

Sulfaguanidine is relatively non-toxic and can be used in the doses described with comparative safety. It is possibly of value in the treatment of ulcerative colitis, but certainly is not a specific, and should be regarded only as an adjunct to the usual forms of therapy.

While the school teacher has not more tuberculosis than the average adult, next to the family she provides the greatest opportunity for close prolonged contact with the school child. To require the teacher to provide a health certificate, including chest films, would serve to remove this reservoir of infection.—D. O. N. Lindberg, M.D., Ill. Med. Jour., Oct., 1935.

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SHALL THE POLITICIANS CONTROL OUR HOSPITALS?

The Social Security Board recommends and President Roosevelt passes it on in his Budget Message that "Social Security benefits be extended at this time to include hospitalization." The President adds the significant statement: "I oppose the use of payroll taxes as a means of war finance unless the worker is given his full money's worth in social security." The clear implication is that war finance is to be used as a cover for an expansion of the Social Security program, thus taking advantage of the national emergency to fasten upon the country a permanent and far-reaching program of state medicine.

Hospitalization is the first step in an all-out program of complete medical care. The demand for the addition of professional services is as certain to follow as night follows day. Our experience with hospitalization insurance fully justifies this prediction. While the Social Security Board may deny the intention, the sequence is so logical as to be inevitable. If this movement succeeds—and it may in a blanket approval of the budget or of the particular section — the medical profession may as well prepare for state medicine in its most objectionable form.

The opposition of the medical profession to state medicine is too well founded and too well known to need repetition. Hospitalization is a real threat to our voluntary system of hospitals which has been developed over a long period of years by private and philanthropic effort. At present, the hospital facilities—notably in the South—are entirely inadequate for a program such as the Social Security Board recommends. Hospitalization provided under the Social Security Act means practically free hospitalization and an invitation to a very large proportion of the population to seek hospital care for minor and chronic ailments that may be safely cared for in the home. The existing hospitals will be crowded beyond their capacity to do efficient work and to provide proper care for the seriously ill, for whom they should be reserved.

The compensation of three dollars a day proposed to be paid to the beneficiary and to the hospitals only on assignment is much below the cost of maintenance under present conditions, and there is, moreover, small chance that the hospitals will get it. This can only mean one of two alternatives—the hospitals must be subsidized by the Government, or the quality of the service must be reduced to the level of an alms house.

The outlook is for increased hospital facilities under governmental control, which means a salaried staff, the restriction of free choice of physician, and a disorganization of the practice of medicine as a free enterprise.

There is at this time less demand than ever before for governmental interference with the normal developments in medical care. Employment and wages are at their peak; farm products are bringing highly remunerative prices; and hospitalization insurance on voluntary plans is meeting with encouraging cooperation of employer and employee. The premium rates are quite within the reach of the larger part of the beneficiaries of social security and all employed persons. Why start a movement for hospitalization at this time, when the Government has about all it can take care of with the most costly war in history on its hands?

THE MORGANTON INVESTIGATION

As the result of a series of newspaper articles by two ex-patients of the State Hospital at Morganton, Governor Broughton appointed a Board of Inquiry to investigate conditions at that institution. This board is composed of five members, and has entered seriously upon the task assigned it. It is apparent that the inquiry will consume much time, but it is to be hoped that the results will justify the time expended.

The irony of the situation is that seven years ago a resolution was adopted by the State Legislature "authorizing the governor to appoint a commission for the study of the insane and mental defectives." A commission of five members, with Dr. Fred Hanes as chairman, was appointed by Governor Ehringhaus. Dr. Lloyd J. Thompson, of the Yale Institute of Human Relations, was employed as Director of the survey, with a staff of three assistants. For a year this group conducted a vastly more thorough and far-reaching study of conditions at all the state hospitals, and at the Caswell Training School, than the present Board can hope to make. In addition, much time and thought were given to various other institutions and conditions that might have a bearing on abnormal mental health in North Carolina. The expense of the survey—amounting to at least $15,000—was generously borne by the Rockefeller Foundation.

Section 3 of the legislature's resolution provided "That the Commission shall, within ten days after the convening of the General Assembly in 1937, make a report to the General Assembly. The report shall consist of the findings of the Commission as to the best methods of care and treatment of the mentally defective and insane persons. The report shall also contain proposed bills. . . . embracing any changes in the present system now in use in North Carolina for the care and treatment of mentally defective and insane persons which the Commission shall deem to be for the best interests of the people of the State of North Carolina."

The Commission faithfully carried out the task assigned it. The study began in October, 1935, and was finished in October, 1936. On December 8, 1936, the report—a volume of 377 pages, including six and a half pages of bibliography—was transmitted to Governor Ehringhaus. One sentence in Dr. Hanes's letter transmitting the report proved to be prophetic: "It is too often the fate of official reports to find themselves quietly interred in the oblivion of dusty pigeonholes." The rest of the paragraph read: "Your Commission earnestly hopes that this report which is so filled with stimulating information, not all of which makes pleasant reading for North Carolinians, will receive the careful attention it so richly deserves."

After six years it may be that this hope will be fulfilled, and that the report will be resurrected from the dusty pigeonholes in which it has slumbered since 1936. Perhaps even yet the generous backing given the Commission by the Rockefeller Foundation may prove to be a good investment. It is rather a sad commentary on human nature, however, that it required these melodramatic stories to reawaken interest in the needs of the mentally afflicted.

* * * *

DEFENSE BONDS

Today our nation confronts the greatest crisis in its history. The situation deeply and intimately concerns the members of the Medical Society of the State of North Carolina, both as practitioners of the art of healing, and as citizens of the United States.

The nation needs us in this war. It needs us as surgeons and physicians for both the armed and civilian forces. It likewise needs us as backers of this all-out struggle to preserve freedom in the world. We are a part of the united civilian army which must provide money for the war effort—money for planes, ships, tanks, and guns, money for ammunition, clothing and equipment for the men in service.

Uncle Sam needs sixty billion dollars this year to meet the bill; maybe next year he will need many billions more. We will pay increased taxes, it is true. A part of the money will be obtained from banks and other lending agencies. But we need more than this—much more, and this must come from the pockets of the people. The Government asks that every American lend dollars through the purchase of Defense Savings Bonds and Stamps.

Not only do Defense Savings Bond dollars pay for tools of war. The dollars so invested are withdrawn from consumer purchasing power, which helps to check inflation. For the investor, the Bonds and Stamps represent individual reserves, just as good as cash in the bank, which can be drawn upon whenever a financial need arises.

The Treasury Department offers three series of Defense Savings Bonds: Series E, F and G. Series E Bonds, "The People's Bonds", can be bought only by individuals. These are appreciation bonds which, if carried through to the 10-year maturity date, provide an average annual return of 2.9 per cent upon the investment. The smallest E Bond costs $18.75 and pays $25 at maturity. Others cost from $37.50 ($50 maturity value) to $750 ($1,000 maturity value). A bond of this series may be registered in the names of one or two persons or in the name of one person with a second listed as beneficiary. All post offices and most banks sell "The People's Bonds".

The F and G Bonds are primarily for associations, corporations, and large individual investors. Series F Bonds, like Series E, are appreciation bonds. They are purchased for 74 per cent of their face value, and at the end of the 12-year maturity period provide a return equivalent to an annual interest rate of 2.53 per cent. The smallest of the F Bonds costs $18.50 and pays $25 in 12 years; the largest costs $7,400 and pays $10,000 at maturity.

The G Bonds, unlike Series E and F Bonds, are sold at par—that is, the cost is the same as the face value. But the G Bonds pay interest semi-annually at the rate of 2½ per cent throughout their 12-year maturity period, thus providing a current income for the investor. Series G Bonds are issued in denominations from $100 to $10,000. Only the Treasury Department and Federal Reserve Banks issue F and G Bonds, but most banks will handle applications for them.

The minimum costs of E, F and G Bonds are $18.75, $18.50 and $100 respectively. But these are not the smallest amounts that can be invested in Defense Savings. Smaller amounts of money purchase Defense Savings Stamps, which range in price from 10 cents to $5.00. When the equivalent of a minimum price bond has been invested in Defense Stamps, they can be turned in for one of the registered interest-bearing bonds.

We, as a united nation, face the future with grim determination. This is total war, and each of us must accept his responsibility for the job to be done. A part of this job is the budgeting of dollars. We must buy and continue to buy the nation's "freedom" Bonds.

* * * *

WHY A MEDICAL MEETING?

With so many demands upon one's time these strenuous days it is reasonable to expect value received for the expenditure of two or three days away from one's professional duties. To be specific, many a "staff room oracle" declares that he is not repaid for attending medical meetings. Some years ago Dr. Henry Christian, in an *Annals of Internal Medicine* editorial, answered the question, "Why a medical meeting?" so effectively that his editorial is reproduced here as a reminder to plan now to attend the annual meeting of the State Society.

"Physicians can be divided into two great groups, those that are learning and those that are forgetting, those that each year know more, and those that each year know less. There seems no third group, those that are stationary.

"A few physicians increase in knowledge from within and grow from their own doing. These are the innate investigators. The rank and file require outside help to grow and to progress. Books, meetings, contacts, discussions, teachers, are our armamentarium for progress. Like the "spring tonic" of past days, all of us need some of this medicine, at least annually, better if it comes more frequently. A large majority of physicians know their need and seek treatment.

"Things in nature rarely are static; they increase or they decrease; they grow or they decay; they progress or they retrogress. Man's education in many respects resembles things of nature; rarely is it static; when knowledge does not increase, almost always it decreases. Physicians should remember this and make every effort to keep out of the static state and on the side of increase, of growth, of progress.

"Contact with colleagues eager to learn, listening to discussions by those capable of teaching, witnessing demonstrations and clinics, seeing scientific exhibits lead to more reading and better observation of patients. **Herein lies medical progress.**"

CASE REPORTS

CLINICO-PATHOLOGICAL CONFERENCE

BOWMAN GRAY SCHOOL OF MEDICINE

Presentation of Case

E. M., a white male aged 35, was first admitted to Vanderbilt University Hospital on November 21, 1930, complaining of stiffness and soreness in the joints. In years past he had experienced migratory joint pains, and four years previously had had pain in the right knee, which was relieved by medicine. Nine weeks before admission practically all of the joints became involved. Swelling and pain were noteworthy; no redness was observed, but he did have low fever. He had remained in bed until he was admitted to the hospital, and had lost 30 pounds in weight.

Physical examination revealed his temperature, pulse, and respirations to be normal except for slight tachycardia. He was obese, and the muscles were flabby. Some limitation of movement was present in both shoulders, but there was no tenderness, redness, or swelling of any of the joints. There was no clubbing of the fingers. The tonsils were enlarged. There was a short apical systolic murmur over the precordium but no other cardiac abnormalities. The urine was clear. The blood was normal except for leukocyte counts of 12,500 and 11,600. A Wassermann test was not performed because the blood specimen was unsatisfactory (hemolysis). The patient was discharged with a diagnosis of chronic multiple arthritis, dental caries, pyorrhea, chronic tonsillitis and obesity.

In the Outpatient Department many teeth were extracted. Blood Wassermann and Kahn tests were negative on January 27, 1931. After January, 1931, the patient was not seen until December, 1933, at which time he reported that he had been well for two years. He returned because of epigastric discomfort, nervousness, a sense of weakness, and a loss of 40 pounds in weight. The heart was not enlarged; the sounds were regular and slow; a blowing apical murmur was present. The blood pressure was 112 systolic, 78 diastolic. Examination of the joints was not remarkable. A fluoroscopic examination of the stomach and duodenum was negative. A stool specimen contained occult blood. A gastric analysis was normal. The patient failed to return after January, 1934, until his second hospital admission on July 19, 1937.

For several weeks prior to this admission he had been unable to work because of shortness of breath, orthopnea, and edema of the ankles. Two years previously (1935) he had developed a penile lesion, which was diagnosed as syphilis, and was treated locally for a time. A few weeks before the onset of his present illness he went to the City Clinic for a blood test, and was informed that he had syphilis. He received two intravenous injections. He had had shortness of breath for about a year.

The blood pressure on admission was 160 systolic, 90 diastolic. It was noted that the patient was a little cyanotic. Frank clubbing of the fingers was present. Slight edema of the ankles and scrotum was present. The peripheral vessels exhibited excessive pulsation, the pulse was collapsing, and a pistol shot sound was heard over the brachial arteries. The point of maximum impulse was not visible or palpable. Right cardiac dullness measured 4.5 by 12 cm.; retromanubrial dullness measured 7 cm. The cardiac rate was rapid; the rhythm regular. The first sound was replaced by a loud, smooth systolic murmur. Along the left sternal border a diastolic murmur was audible—loudest at the third left interspace but clearly heard over the right second interspace, where it was associated with a louder systolic murmur. There were moist rales over the lung bases; liver dullness was not increased, and the liver was tender. The urine and routine blood examinations were not remarkable. The Wassermann and Kahn tests were positive. The blood examination was negative. The cerebral spinal fluid contained 4 cells; colloidal mastic and Pandy tests were negative. The cerebrospinal fluid Wassermann test was positive in all dilutions. An electrocardiogram revealed slurred QRS complexes and depressed ST segments; no left ventricular preponderance was noted.

An x-ray of the chest showed the following heart measurements: from the midsternum to the right border 6.2 cm.; from the midsternum to the left border 11.2 cm.; aortic arch, 8.8 cm. There was extensive fibrosis and evidence of passive congestion in the lower lobes, and some fibrosis in the right upper lobe.

The patient was given diuretics and was

digitalized, and he improved greatly. Upon discharge he was instructed to restrict fluids and was placed on bismuth and potassium iodide therapy and maintenance doses of digitalis.

He continued to visit the Outpatient Department, and received bismuth, potassium iodide, and numerous treatments with salyrgan. His weight varied between 160 and 190, depending upon the amount of edema present. Steadily decreasing cardiac reserve finally forced him into the hospital for his third and final admission, on May 12, 1938.

On this admission Cheyne-Stokes respiration was present. The eyes were puffy, the superficial veins full. There was marked orthopnea and marked edema of the lower extremities and back. He was a little cyanotic. Well marked clubbing of the fingers was present. Edema of the retina was observed. Rales were present at the bases of the lungs, and a rather large, tender liver was palpable. The heart signs were essentially as they had been on the previous admission except that the organ appeared to be larger. A systolic murmur was heard over the carotids. The blood pressure was 150 systolic, 70 diastolic. The urine contained albumin (1 plus) but no casts or red blood cells. The Wassermann test was positive. The nonprotein nitrogen was 50 mg. per 100 cc. The stool examination was negative for occult blood. Toward the end of the first week in the hospital—during which time his temperature rose to 100 or 101 F. in the evenings—the patient developed pain over the left chest which was intensified by breathing and coughing. Three days later evidence of apparent consolidation of the left lower lung was noted. The white blood cell count was 13,200. Evidence of congestive failure increased during the second week, and the patient responded poorly to salyrgan therapy. Fever continued, and the leukocyte count was 20,000. On the day of death, May 26, he passed several stools which contained gross blood. Respirations became increasingly difficult and irregular, and finally ceased.

Discussion

DR. TINSLEY R. HARRISON: It is clear, from the record, that the most important site of disease in this patient was the heart. The chief problem is the determination of the type of cardiac disease. It is well to approach all cases in which there are disorders of the heart from three standpoints: the functional, the anatomical and the etiological. As regards cardiac function, we may say that this patient had advanced and severe congestive heart failure, which was the chief factor in producing death. Anatomically, we can say that the patient had enlargement of the heart, due no doubt to a combination of dilatation and hypertrophy, and had some dilatation of his aorta. It is also quite clear that the patient had a well marked aortic regurgitation. At this point certainty ceases and we enter the realm of speculation. Did he have stenosis as well as insufficiency of the aortic valve? He lacked some of the classic signs of aortic stenosis, but he had at least a moderately loud systolic murmur at the base which was transmitted into the vessels of the neck. This finding, taken with the rest of the picture, makes me believe it probable that he did have early aortic stenosis. As regards the mitral valve the evidence, while not conclusive, is strongly suggestive of an organic lesion. Had the systolic murmur at the apex appeared late in the disease, one could readily ascribe it to dilatation resulting from disease of the aortic valve. However, the apical murmur came first, and in a patient with his history it seems highly probable that there was organic disease of the mitral valve. Since signs of stenosis were not found, it would appear that the mitral lesion was considerably less advanced at the time of death than the aortic lesion.

The next problem is the difficult one of etiology. Often in medicine we lack a clue as to the etiology of a given condition. Here our problem is quite different. We have so many clues that their very abundance is confusing. Since the patient's first complaint involved the joints one naturally thinks first of rheumatic heart disease. The story points strongly toward rheumatic fever, and indicates not one but several attacks. If such is the case, the statistical probabilities of valvular heart disease on a rheumatic basis are very strong. One can safely predict that something like 80 to 90 per cent of the people who have had repeated attacks of rheumatic fever will eventually develop endocardial disease. Furthermore, the first evidence of any valvular disease in this patient was in the mitral valve, and although the lesion here was not very progressive,

the fact that the apical systolic murmur came first constitutes very strong evidence that the initial valvular disease was of rheumatic origin. Finally, if we accept the signs at the base as indicating early aortic stenosis, we are forced to assume, in a man of this age, that there is rheumatic disease of the aortic valve. In older patients one sometimes has stenosis as a result of calcium deposition, even in the absence of antecedent rheumatic fever, but in a man this age rheumatic fever is practically the only cause of aortic stenosis. Syphilis, which is a common cause of aortic regurgitation, does not cause aortic stenosis. Hence, before we attempt to evaluate the significance of syphilis I think we have to conclude that this patient had rheumatic disease of his mitral valve, not far advanced, and rheumatic disease of his aortic valve at least moderately advanced.

To say that the main lesion of the heart valves is rheumatic is not to deny that syphilis may play a role. The two conditions may occur in the same heart and, indeed, I have twice in the past seen this at autopsy. Even though there was evidence of rheumatic heart disease prior to the acquisition of syphilis, there are several points which suggest that the patient may later have developed cardiovascular syphilis. Chief among these is the width of the aortic shadow in the x-ray. Granting that a widened aortic shadow does not necessarily mean syphilis, and that it may occur as a result of hypertension or arteriosclerosis, and that some widening may be observed in patients with rheumatic disease of the aortic valve, it still seems probable that an aortic shadow 9 cm. wide in a patient known to have syphilis indicates luetic aortitis. I am therefore going to make a diagnosis of syphilitic aortitis and will leave the question open as to whether syphilis involved the aortic valve too. I think the main lesion in the aortic valve was rheumatic, but if the man had syphilitic aortitis, it is probable that there was some syphilitic involvement of the aortic valve as well.

However, we still have something else to account for in regard to this patient's heart. He had clubbed fingers, and these appeared while he was under observation. Since at the earliest examination he did not have clubbed fingers, we can exclude the possibility that he was one of those rare individuals who happen to have this sign without any pathological process to account for it or that congenital heart disease was the cause of the clubbed fingers. The patient had throughout his illness certain signs in his lungs which cannot be readily explained on the basis of congestion alone, and hence one might think that the pulmonary disease was the cause of the clubbed fingers. Against this is the fact that even after the clubbed fingers appeared cyanosis was quite slight. Usually, when a person has clubbing of the fingers as the result of chronic pulmonary disease, there is also well-marked cyanosis. Hence, I do not believe that the clubbed fingers can be explained on a pulmonary basis. Under exceptional circumstances patients with cirrhosis of the liver may develop clubbed fingers, but this patient had no evidence of such a disease. Occasionally one gets clubbing in an individual with an aneurysm. However, this man's syphilis was quite recent, and it seems very improbable that the disease had endured long enough to give him an aneurysm. Hence, by exclusion, we are forced to give serious consideration to that form of heart disease which commonly causes clubbed fingers in the absence of any striking cyanosis—namely, bacterial endocarditis. Certainly, the combination of rheumatic heart disease and clubbing of the fingers developing under observation suggests this diagnosis very strongly. On the other hand, the patient lacked some of the classical manifestations of bacterial endocarditis. No mention is made of fever and no petechiae or other embolic manifestations are described. Therefore, we have to conclude either that he did not have bacterial endocarditis, or that it existed in an inactive form. On the whole the latter conclusion seems to me to be more likely, and I am going to make a diagnosis of bacterial endocarditis in the healed or bacteria-free stage.

But this immediately raises another question. What about the kidneys? The patient had slight nitrogen retention (but not more than could be accounted for by congestive heart failure), and a little albumin in the urine. Neither of these findings is particularly significant in a person known to have heart failure. However, there is a close association between bacterial endocarditis and nephritis. Patients in the active stage of the disease, during which bacteria are present in the heart valves and can be recovered from the blood stream, commonly have the

so-called embolic nephritis, which might better be called "multiple glomerular embolization." Patients with healed bacterial endocarditis or individuals in the bacteria-free stage of the disease have an entirely different type of nephritis, which resembles the ordinary glomerular nephritis. This is such a common finding that the diagnosis of healed bacterial endocarditis almost automatically forces one to consider the probability of glomerular nephritis.

Shortly before death the patient developed fever and pain in his chest with signs which were interpreted as consolidation of the left lower lobe. Possibly he had a terminal acute pneumonia, but from the description I think it much more probable that this was an infarct of the lungs.

As regards the bloody stools which were passed in the last stage of the illness there are several possibilities, the most likely being that he had either a simple terminal colitis, such as one commonly sees in moribund patients, or mercurial colitis due to the diuretic drug. Since the former is such a common occurrence I think it the more likely condition.

In summary, then, I am inclined to believe that we are dealing here with three types of cardiovascular disease occurring in the same patient: chronic rheumatic heart disease affecting the mitral and aortic valves, a superimposed bacterial endocarditis which is healed, and syphilitic aortitis. In addition, I would expect the patient's organs to show an infarct in the lungs, chronic glomerular nephritis, and an acute terminal colitis.

Dr. Harrison's Diagnosis

Rheumatic heart disease affecting the mitral and aortic valves.
Healed bacterial endocarditis.
Syphilitic aortitis.
Pulmonary infarction.
Chronic glomerular nephritis.
Terminal colitis.

Anatomical Report

DR. ROBERT P. MOREHEAD: The heart was markedly enlarged and weighed 850 Gm. All chambers were involved in the process, but there was a disproportionate enlargement of the left ventricle. The mitral valve measured 9 cm. in diameter and was characterized by a diffuse and generalized thickening of the cusps; a few small nodular vegetations were seen on the surface. The chordae tendineae were shortened and thickened. It was evident that the valve was both stenosed and incompetent. Microscopically there was marked fibrosis and perivascular round cell infiltration. The aortic valve was reduced to 6 cm. in diameter. The left cusp was almost completely destroyed and the posterior cusp showed marked thickening with rolling of the free edges. The right cusp was thickened and distorted and two distinct points of perforation were present. Immediately above the left cusp was a small pocketing of the aorta, and a similar pocket was seen on the opposite side above the right cusp. Stellate and longitudinal wrinkling of the vessel was evident. Microscopically the picture was that of fibrosis, necrosis, scarring, diffuse plasma cell infiltration and perivascular mononuclear infiltration.

It was evident from the gross and microscopic findings that this patient suffered from inactive rheumatic endocarditis, involving both mitral and aortic valves, resulting in mitral stenosis and insufficiency and aortic insufficiency. The gross and microscopic features of the aortic valve also suggested a healed bacterial endocarditis. There was also gross and microscopic evidence of syphilitic aortitis and valvulitis. A few vegetations were seen on the tricuspid valve, but the valve was normal in size and thickness.

The viscera displayed evidence of marked chronic passive congestion. The liver was markedly enlarged and presented a typical "nutmeg" appearance. The lung showed brown induration and fibrosis was fairly well marked. It is difficult to eliminate healed rheumatic interstitial fibrosis in this case and to attribute all of the fibrosis to chronic passive congestion. In the right lower lobe of the lung a large infarct was seen and a large adherent thrombus was found in the lower branch of the right pulmonary artery. The spleen was enlarged and engorged with blood, and the intestines showed vascular engorgement with extravasation of blood into the lumen of the bowel.

Other findings of interest were those of gastric and intestinal hemorrhages with minimal ulceration and secondary infection. The pancreas showed chronic interstitial inflammation and the kidneys were the seat of chronic glomerular nephritis.

This case is interesting in that it presents in a single patient the three principal types of valvular disease, and that all of the clinical data are readily explained from the pathological findings.

Anatomical Diagnosis

Rheumatic heart disease involving the mitral and aortic valves with minimal tricuspid involvement.

Healed bacterial endocarditis, aortic valve.

Syphilitic aortitis and valvulitis.

Chronic interstitial pneumonitis, rheumatic.

Pulmonary thrombosis and infarction.

Chronic glomerular nephritis.

Enteritis.

Chronic pancreatitis.

Chronic passive congestion of all viscera.

MEDICOLEGAL ABSTRACT

J. F. Owen, M.D., LL.B.
Raleigh

Insane Persons: In proper instances the clerk of court, with the approval of a Superior Court Judge, may order the purchase of a home for the use of certain dependents of incompetents, and may order proper advances made for support.

This is an account of a special proceeding heard before the Clerk of Superior Court of one of our eastern counties, he at the time acting in his judicial capacity. As is known, clerks of court are clothed with authority to try certain cases having to do with the estates of decedents, orphans' funds, estates of incompetents, and other matters of a similar nature. They have the usual duties in this respect which were formerly held by probate judges. In this particular instance the matter coming to the attention of the clerk of court was somewhat unusual in that the petitioner, who requested a sufficient amount of money from the estate of an incompetent person to purchase a house for her use and also a certain amount of money to be awarded her monthly for her support, was not the wife or a minor child of the incompetent. At the hearing the presiding judge of the Superior Court of the county sat with the clerk during this trial and heard the evidence and arguments of counsel, after which the judge approved the action of the clerk. The facts in this case as shown by the testimony are as follows: The petitioner was a sister of a World War Veteran who at the time of this trial was considered incurably insane and was a patient in a government hospital, where he was being cared for without charge. It was proved satisfactorily to the court that the veteran gave his sister all the assistance he could for her support before he was drafted and that he sent her money for this purpose after he was in the army. Evidence also showed that he was unmarried and had no dependents. When the petitioner instituted this special proceeding the incompetent had in the hands of his guardian, a bank, the sum of $2,246.81, representing payments on his War Risk Insurance. Testimony indicated that his sister was destitute and without means of support. After the evidence was in and the arguments of the counsel heard before the clerk and the aforementioned judge, the clerk ordered that the home be purchased for the petitioner and that the sum of $20 monthly be allowed as a reasonable amount for her support. The following aspect of the case has no medicolegal significance, but it is perhaps of some general interest. The clerk ordered that for "valuable services" of attorneys the sum of $625 be allowed for attorneys' fees. This judgment was appealed to the Supreme Court by the guardian, and the appellate court held as follows: "The clerk of court, with the approval of the resident or presiding judge, has the power upon the proper findings from the evidence to order the guardian to purchase a home in the name of the incompetent for the use of the petitioner and to advance the petitioner a reasonable sum monthly for her support. The attorneys' fees however, are not a part of the cost and may not be recovered by the successful litigant."

The basis for the above decision of the Supreme Court with reference to the award of a home and a certain amount of money for the petitioner's support is contained in a part of Section 2296, N. C. Code, and is as follows: "Whenever any non-sane person of full age, not being married and not having issue, be possessed or his guardian be possessed for him, of any estates, real or personal, or of an income which is more than amply sufficient to provide for such person, it shall be lawful for the Clerk of the Superior Court for the county in which such person resided prior to insanity to order from time to time, and so often as may be expedient, that fit and proper advancements be made, out of the surplus of such estate or income, to his parents, brothers, and sisters, or grandparents to whose support, prior to insanity, he contributed in whole or in part." The above, of course, has nothing whatsoever to do with the findings as to attorneys' fees, this being entirely incidental. (Vol. 216, p. 525, North Carolina Supreme Court. Decision rendered spring term, 1939.)

Cerebral Thrombosis.—In older persons who suddenly fail in health, one should think always of the possibility that there has been a small cerebral thrombosis. This is a common lesion which in my experience is seldom diagnosed until after the patient has had one or more typical strokes with involvement of arm or leg or speech center. In my youth I often watched these patients die by inches; I did not know what was the matter, and my colleagues did not seem to either. It was often thought the trouble was in the heart, but it was not. The patient often came in with what he called stomach trouble. Why? Because, suddenly, at a certain minute of a certain day there came a distress in the abdomen. Perhaps the patient was dizzy and vomited and for an hour or two was confused. The thing that always impresses me in these cases and makes the diagnosis for me is the suddenness of the onset. Highly diagnostic, also, when it is present, is the big change in the patient's character.—Walter C. Alvarez: What Is the Matter With the Patient Who Is Chronically Tired? J. Missouri State M. Assoc. 11:367 (November) 1941.

MILITARY MEDICINE

March 5, 1942.

Dear Dr. Johnson:

I have been designated as State Procurement Officer by Major Samuel F. Seeley, Executive Officer and Paul McNutt, Chairman of the Procurement and Assignment Service of the Defense Health and Welfare Services in Washington, D. C. The purpose is for the Procurement and Assignment of Physicians, Dentists, and Veterinarians.

Our Medical Committee has to deal with the Procurement and Assignment of Physicians. I have been asked to set up a Central Committee. The following is a list of those who have accepted this assignment:

Dr. Fred Patterson, Greensboro
Dr. E. J. Wannamaker, Charlotte
Dr. Roscoe McMillan, Red Springs
Dr. Carl V. Reynolds, Raleigh
Dr. W. D. James, Hamlet
Dr. Leslie Lee, Kinston
Dr. William Coppridge, Durham
Dr. R. P. Noble, Raleigh
Dr. Douglas Craig, Winston-Salem
Dr. Charles C. Orr, Asheville
Dr. J. R. McCracken, Waynesville
Dr. Webb Griffith, Asheville
Dr. Donnell Cobb, Goldsboro
Dr. James Robertson, Wilmington

Each County Society under Dr. Webb Griffith, President of the North Carolina Medical Society, is to set up a County Committee. The function of the whole group is to pass on the availability of any physician for service in the United States Army whose name is sent to the State Chairman and in turn sent to the member of the Central Committee in whose locality the physician resides and also to the County Committee.

I am enclosing a copy of a letter I received yesterday which is self explanatory and which you may publish in full if you so desire.

With best wishes, I am

Sincerely yours,
Hubert B. Haywood, M.D.
State Chairman
Procurement and Assignment Service

* * *

February 27, 1942.

Brigadier General Vivian Collins
State Director for Selective Service
State Arsenal
St. Augustine, Florida

Dear General Collins:

The following telegram from Major Sam F. Seeley, Executive Officer, Procurement and Assignment Service, Defense Health and Welfare Services, 601 Pennsylvania Avenue, Washington, D. C., received today:

"Urgently recommend you contact State Directors Selective Service System regarding the problem which the Procurement and Assignment Service is facing as a result of the classification by Local Boards of physicians, dentists and veterinarians in Class I-A with view of induction as enlisted men. Direct their attention to Selective Service System Memo 1-363 January 28, 1942, reproduced page 633 Journal American Medical Association February 21, 1942. Present strong representations that all physicians, dentists and veterinarians will be enrolled with Procurement and Assignment Service within next few weeks and that overall needs of nation cannot be met unless all physicians, dentists, and veterinarians are retained in their professional capacities. Since we are responsible for the utilization of these individuals throughout the nation Local Boards should look to us for their assignment even if at present excessive in local areas; also at such time as those not willing to serve in professional capacities through the Procurement and Assignment Service are determined, they will be referred back to the Local Boards for final disposition."

It is urgently requested that you call attention of each Local Board in your State to this situation in order that the services of practitioners of medicine, dentistry, and veterinary medicine may be given the opportunity to practice the profession for which they are best qualified, either in a community requiring their services or as commissioned officers with the armed forces.

Each Local Board is respectfully requested to grant the necessary deferment of any physician, dentist or veterinarian in the above category who is necessary as a practitioner of his profession in a community or who desires service with the armed forces in order that he may have sufficient time to obtain a commission or placement through his enrollment with the Procurement and Assignment Service.

Yours very truly,
Edgar H. Greene, M.D.
Chairman, Committee for
Procurement and Assignment,
Fourth Corps Area

March 17, 1942.

Dear Dr. Johnson:

Governor J. M. Broughton has asked me to serve on the Salvage for Victory Campaign in North Carolina as a representative of the North Carolina Medical Society.

The North Carolina Medical Society and all hospitals are asked to participate in this campaign. We are asked to salvage paper, rags, rubber, and metals. There is a definite shortage and it is important to our war effort that we husband every resource. It is estimated that ten pounds of scrap metal will make twenty-five pounds of armament for our soldiers. Scrap metal is a necessary ingredient of steel. It is estimated that there are over eighty million pounds of scrap metal on the farms of North Carolina. The Japanese created a billion dollar a year industry in the Southeastern United States. Charitable organizations or individuals after collecting salvage materials may dispose of it to junk dealers. During the month of February twenty-eight million pounds of scrap metal was salvaged in North Carolina. The Government has a record of all old scrap cars in the "auto-graveyards" and can claim them at their own price at any time. There is at present a shortage of freight cars for hauling this metal. There are twenty-eight steel mills in the United States that are using this scrap metal.

It is estimated that in addition to the sugar shortage there will eventually be a shortage of glycerine. It is interesting to note that it takes 1/5 of an acre of sugar beets to produce enough of the ingredients of the explosive needed to fire a sixteen inch gun one time.

We of the Medical Profession are therefore asked to help salvage these materials for an American victory.

Very truly,
Hubert B. Haywood, M.D.
Member Salvage for Victory Campaign

First Aid For Air Raid Casualties

First aid measures applicable to the emergencies of war are embodied in a "Handbook of First Aid" issued by the Medical Division of the Office of Civilian Defense in cooperation with the American National Red Cross for the use of enrolled civilian defense workers.

The new handbook is of pocket size and has twelve chapters titled as follows: Advice to the Civilian Defense First Aid Workers; General Considerations; Care of Wounds; Dressings and Bandages; Hemorrhage; Burns, Shock (Collapse); Fractures; Artificial Respiration; Transportation of the Injured; Chemical Warfare; Miscellaneous Conditions. It will be seen from the titles that all material not relevant to the war emergency has been omitted from consideration. The book does not replace the standard Red Cross "Textbook of First Aid", which is to be used in first aid classes.

Textbook On War Gases

"Protection Against Gas," a textbook on war gases to be used for instruction of public employees and volunteers enrolled in the various groups of the United States Citizens' Defense Corps, has been issued by the U. S. Office of Civilian Defense. The book was prepared by the War Department under the direction of the Chief of Chemical Warfare Service, U. S. Army, with suggestions of the National Technological Civil Protection Committee, a special advisory committee of engineers. The section on "First Aid Treatment of Gas Casualties" was prepared with the assistance of the Medical Division of OCD.

BULLETIN BOARD

SECRETARY'S MESSAGE

Eighty-Ninth Annual Session
Medical Society of the State of
North Carolina
Charlotte, N. C.
May 11, 12 and 13, 1942

Plans are rapidly shaping up, and, from all indications, we will have an outstanding meeting. Headquarters will be the Hotel Charlotte, a hotel with excellent accommodations. The following is a list of the hotels in Charlotte:

Hotel Charlotte:
Single Rooms—$3.30 to $5.50
Double Room (Double Bed)—$4.50 to $7.00
Double Room (Twin Beds)—$5.00 to $8.00

Barringer Hotel:
Single Rooms—$3.25
Double Rooms (Twin Beds)—$5.00

Selwyn Hotel:
Single Rooms—$2.25 to $3.00
Double Rooms (Twin Beds) with Bath—$4.00 to $5.00
Double Rooms (Twin Beds) without Bath—$3.00
Single Room without Bath—$1.75

Mecklenburg Hotel:
Single Rooms—$1.75 to $3.00
Double Rooms (Double Bed)—$3.25 to $4.00
Double Rooms (Twin Beds)—$4.00

Mayfair Hotel:
Single Rooms—$2.00 and $3.50
Double Rooms—$3.00 to $4.50

I suggest that you make your reservations as early as possible, for I have been from one end of the state to the other attending various meetings and have good reason to believe that there will be a record attendance and that hotel accommodations will be at a premium. Requests for reservations should be addressed to the management of the hotel of your choice or to Dr. Harry Winkler, 121 West Seventh Street, Charlotte, who is Chairman of the Hotel Committee.

Naturally, the theme of this meeting will be centered around the war, but I promise you three full days of fellowship, frivolity and fun as well as an opportunity to increase your store of professional knowledge and to learn just what may be expected of you as your professional contribution toward the winning of the war. Let us take full advantage of this meeting. Come early and stay with us for "Auld Lang Syne". There will be enjoyable social features, strong professional stimuli, and a demonstration that we subscribe to the motto "E Pluribus Unum" professionally as well as nationally. Bring your colleagues from your county society and let us see if we can arouse some good-natured rivalry in winning the honor of having the best attendance (percentage) among the component county societies.

As for our Exhibits, all available space has been taken for Commercial Exhibits, and our Scientific Exhibits are far above the average. For instance, Major O. C. Paciulli, Commanding Officer, Ninth Medical Battalion, Fort Bragg, will have an exhibit showing how 400 casualties may be taken care of at once in a Field Hospital. This, in itself, will be well worth the trip to Charlotte. The Scientific Exhibits will fill the largest space we have ever had for such exhibits.

The House of Delegates will convene at 2 p.m. on Monday, May 11, in the Ball Room of the Hotel Charlotte.

At 7:30 a.m., Tuesday, May 12, at the Hotel Charlotte, we are initiating an innovation, a Breakfast for the Presidents and Secretaries of the component county medical societies. This is being instituted in the hope of stimulating a fuller relationship between the officers of the State Society and those of

the county societies. We have planned a very interesting and instructive program. There will be three or four speakers, who will each talk for only a few minutes. Their talks will be followed by a round-table discussion of the various problems encountered by the officers present. I urge each President and each Secretary of a county medical society to back me up in this venture by being present with his problems and his ideas. As a result of this meeting together I hope that each of us will be able to carry home an inspiration to work more closely together because of a better understanding with one another.

At the General Session on Tuesday morning, May 12, Dr. Fred Rankin, President-Elect of the American Medical Association, who is now in the office of the Surgeon General in Washington, will talk to us on "The Medical Profession and Mobilization". Another headliner for the First General Session will be Brigadier-General H. C. Coburn, Commanding Medical Officer at Fort Bragg, who will talk on "The Responsibilities of the Civilian Physician in the War Emergency".

Tuesday at noon there will be Alumni Luncheons, followed in the afternoon by various sectional meetings. The chairmen of these sections have completed their programs and each has done very fine work in securing outstanding speakers.

Tuesday evening will be "President's Night". The President's Dinner, to be held at 7 o'clock in the Ball Room of the Hotel Charlotte with Dr. Oren Moore as Toastmaster, is an event you must not miss. Incidentally, for this occasion, you may leave all your trials, troubles and tribulations in your rooms, for we have secured an extraordinarily inspirational and humorous speaker, Mr. James E. Gheen of New York City, who will be the only speaker of the evening. As a philosophical humorist, he has no peer on the public platform. At 10 o'clock the President's Ball will begin. Bring along your dancing shoes so that you may fully enjoy this feature of our entertainment.

On Wednesday morning, May 13, the Second General Session will begin in the Ball Room promptly at 9 o'clock. When I tell you that Dr. Paul Dudley White will be the guest speaker, enough is said. In addition to this attraction, your program is brim-full of well worth while talks by outstanding North Carolina physicians. We will begin on time and end on time and WOE unto the fellow who oversteps his limit! This program is being timed to the minute.

Other Alumni Luncheons will be held on Wednesday at noon, and the sectional meetings which will follow in the afternoon are far above par.

The program for the Woman's Auxiliary, under the presidency of Mrs. Sidney Smith of Raleigh, will begin at 1:30 p.m. on Monday, May 11, with a Luncheon for the Executive Committee and the Board of Directors, courtesy of Mrs. Smith, at the Hotel Charlotte. From then on there is something to be done every minute. On Tuesday, Mrs. Roscoe E. Mosiman of Seattle, Washington, President of the Woman's Auxiliary to the American Medical Association, will be the guest speaker.

There will be a visit to the Mint Museum of Art, followed by a Coffee Hour at the home of Mrs. J. A. Elliott. A visit to the beautiful gardens of Charlotte will be followed by Tea at the home of Mrs. E. J. Wannamaker, wife of the President of the Mecklenburg County Medical Society. There will be various other attractions which the ladies of the Charlotte Auxiliary have arranged.

The North Carolina Public Health Association, which heretofore has held its meeting on Monday preceding the meeting of the Medical Society, this year will have two full days beginning Thursday morning, May 14, with a General Session, the program for which should be both interesting and instructive. There will be another interesting General Session in the afternoon. Mr. Edwin Gill of Raleigh, North Carolina Commissioner of Paroles, will speak on the subject "Diseases and Crime" at the Annual Dinner to be held at Hotel Charlotte on Thursday evening, May 14.

The Sectional Meetings of the Public Health Association on Friday, May 15—the Health Officers' Section, the Nurses' Section and the Sanitarians' Section—will make up a full day.

The multitudinous details necessary for the preparation and arrangements for the State Meeting have largely been attended to by the different committees of the Mecklenburg County Medical Society, headed by Dr. Joseph A. Elliott as General Chairman, and much of the success and smoothness of this work is creditable to the prideful contribu-

tions in effort and plans of the Mecklenburg County Society as a whole.

Remember to be in Charlotte on May 11, 12 and 13.

* * *

Since the above was set in type, it has been necessary to rearrange Wednesday's program because of conflicting engagements of Dr. Paul Dudley White. Dr. White is scheduled to address the New Hampshire Medical Society at Manchester on Tuesday. The New Hampshire Society has rearranged its program so that he may come to Charlotte. In order to secure Dr. White we think it well worth while to rearrange our program. Therefore, instead of the second General Session's being held on Wednesday morning, as has been the case in the past, it will be held on Wednesday afternoon in the ball room of the Hotel Charlotte, and the section meetings heretofore scheduled for Wednesday afternoon will be held on Wednesday morning.

News Notes From Duke Hospital

In order to cooperate with the accelerated medical school program, the Duke Hospital internships of twelve months in the various services will commence on July 1, 1942, April 1, 1943, January 1, 1944, October 1, 1944, and July 1, 1945. This schedule will provide an overlapping in internships for a period of three months, during which the preceding group will be senior interns. Applications should be sent to the Superintendent six months before these dates.

News Notes From the Bowman Gray School of Medicine of Wake Forest College

Mr. Charles Dollard, Assistant to the President of the Carnegie Corporation of New York, visited the school on March 20, 1942.

Dr. C. Nash Herndon has returned to the school from the University of Michigan to resume his activities in the Department of Medical Genetics. Dr. Herndon was Research Associate in the Department of Human Heredity of the University of Michigan while on leave from this medical school.

Dr. Camillo Artom, Dr. W. H. Fishman and Miss Marjorie Swanson of the Department of Biochemistry, Dr. Herbert S. Wells and Dr. J. Maxwell Little of the Department of Physiology and Pharmacology, and Dr. Arthur Grollman, Research Professor of Medicine, attended the meeting of the American Federation of Biological Sciences held in Boston.

Dr. Roland E. Miller, Assistant Professor of Anatomy, attended the meeting of the American Association of Anatomists held in New York City.

Dr. Howard H. Bradshaw, Professor of Surgery, will give a series of ten lectures on the treatment of chest wounds to the local physicians in Winston-Salem who anticipate going into the service. These lectures begin on April 1.

News Notes From the University of North Carolina

Dr. William deB. MacNider attended a meeting of the American Division of the Club for Research on Ageing in Baltimore February 28 to March 1. At this meeting Dr. MacNider was reelected Chairman of the American Division of the International Club for Research on Ageing.

* * *

Dr. Milton J. Rosenau and Dr. Harold W. Brown, of the faculty of the School of Public Health, attended the meetings of the American Epidemiological Society and the Association of Schools of Public Health in Baltimore on March 20 and 21, and the meetings of the State and Provincial Health Authorities with the Surgeon General of the U. S. Public Health Service in Washington on March 22, 23, and 24. Dr. H. G. Baity, of the faculty of the School of Public Health, also attended the meetings of the State and Provincial Health Authorities in Washington.

* * *

Dr. William L. Fleming, of the faculty of the School of Public Health, was in Washington on March 23 and 24 to attend a meeting with the National Research Council.

News Notes From the State Board of Health

Dr. Carl V. Reynolds, North Carolina State Health Officer, was elected President of the State, Territorial and Provincial Health Authorities of North America, at the annual meeting of that organization held in Washington.

Dr. Reynolds succeeds Dr. Frederick W. Jackson, Deputy Minister of Health at Winnipeg, Manitoba, who served as president during the past year.

Other officers elected were: Vice-president, Dr. Gregoire F. Amyott, Provincial Health Officer, Victoria, British Columbia; secretary and treasurer, Dr. Albert J. Chesley, State Health Officer of Minnesota, who was reelected for his eighteenth term.

New members elected to serve on the executive committee were: Dr. Edwin S. Godfrey, Jr., State Health Officer for New York; Dr. Carter W. Williams, State Health Officer for Tennessee, and Dr. Donald G. Evans, Health Officer for the State of Washington.

A new organization was formed, composed of State and territorial health officers of the United States and its possessions, to act as a clearinghouse on matters of federal import relating to the various States and territories.

The work of this organization, said Dr. Reynolds, who was president protem, is to review health problems of an interstate nature and to coordinate the interests of all concerned.

* * *

Of the 446 babies under a year old who died in North Carolina during February, 18 smothered to death while sleeping with parents, according to reports made to the State Board of Health. About 75 such deaths occur in the State annually, most of them in wintertime.

February deaths from influenza totaled only 48, as compared with 291 the corresponding month in 1941, while pneumonia deaths for the month dropped from 322 to 231. Measles claimed 11 victims, against 2 in February last year. From January 1 through March 14, 1942, there were 10,865 cases of measles reported in North Carolina, against 3,243 during the corresponding period of last year and 1,070 in 1940. The disease, on the up-surge this year, is scattered throughout the state.

Suicides are temporarily on the decrease, there having been only 15 reported last month, against 25 in February, 1941, while homicides for the month totaled 24. There were 28 reported in February last year.

Reports for the year through February show that an average of 235 babies were born in the State each day, while daily deaths averaged 89.

North Carolina Neurologic and Psychiatric Association

The North Carolina Neurologic and Psychiatric Association met in Charlotte on March 27. Dr. B. J. Alpers, Professor of Neurology at Jefferson Medical College, was the guest speaker for the dinner meeting. His subject was "The Structural Basis of Psychiatry". Others appearing on the program were Drs. Paul Kimmelstiel, Archie A. Barron, W. B. Mayer, and D. G. Welton of Charlotte; Drs. Robert Graves, Ignacio Matte, and Maurice H. Greenhill of Durham; Dr. Fred Taylor of High Point; Dr. James Watson of Raleigh; Dr. Willard Cardwell of Greensboro; and Dr. J. A. Rose of Winston-Salem. Officers for the past year were Dr. Archie A. Barron of Charlotte, President; Dr. F. B. Watkins of Morganton, Vice-President; and Dr. Burke Suitt of Durham, Secretary and Treasurer.

Cancer Control Institute

The Woman's Field Army of the American Society for the Control of Cancer held a Cancer Control Institute in Winston-Salem on March 24, Mrs. George E. Marshall, State Commander, presiding. Physicians appearing on the program of the dinner meeting were Dr. H. B. Ivey, Chairman of the State Executive Committee; Dr. Ivan M. Proctor, Educational Director; Dr. F. Webb Griffith; and Dr. C. C. Carpenter, Dean of the Bowman Gray School of Medicine of Wake Forest College.

April is national cancer control month.

Buncombe County Medical Society

Dr. Charles Henry Armentrout addressed the Buncombe County Medical Society on March 16. His subject was "Potassium Thiocyanate in the Treatment of Hypertension".

Forsyth County Medical Society

The Forsyth County Medical Society held a dinner meeting in Winston-Salem on March 10. Dr. Barnes Woodhall of Duke University School of Medicine spoke on "Rupture of Intervertebral Discs".

Haywood County Medical Society

The March meeting of the Haywood County Medical Society was held in the Haywood County Hospital on March 19. Dr. Dudley Smith of Waynesville gave a talk on his experiences with the American Hospital in Britain, where he has served during the past year. The dentists and nurses were invited to attend this meeting.

American College of Physicians

The Twenty-Sixth Annual Session of the American College of Physicians will be held in St. Paul, Minnesota, April 20-24. Dr. William deB. MacNider of the University of North Carolina School of Medicine will deliver the Convocational Address at the Annual Convocation to be held Wednesday evening, April 22. His subject will be "A Consideration of the Factor of Change in the Animal Organism". Others appearing on the program from North Carolina are Dr. Verne S. Caviness, Dr. Thomas L. Umphlet, and Dr. Chauncey L. Royster of Raleigh, who will give a paper on "Blood Pressure and Sulfocyanates"; and Dr. C. H. Cocke of Asheville, who will hold a clinic on "Pulmonary Conditions: Atelectasis and Spontaneous Pneumothorax".

Planned Parenthood Federation of America

Dr. Claude C. Pierce, until March 1st a Medical Director of U. S. Public Health in charge of District No. 1, will assume active duties as National Medical Director of the Planned Parenthood Federation of America, Inc., it was announced by Richard N. Pierson, President of the organization, which was formerly the Birth Control Federation of America, Inc.

News Notes

Dr. W. R. Pitts and Dr. William Francis Martin of Charlotte have recently been certified by the American Board of Surgery.

* * *

Dr. Robert T. Odom has opened offices in the Nissen Building Winston-Salem, for the practice of surgery.

* * *

Dr. Thomas W. Baker of Charlotte was married to Miss Margaret Lunsford in Durham on March 29.

The Japanese death rate is 17.4 per 1000 as compared to 10 or 11 in the United States. Japan's present death rate, in fact, resembles ours of 1900. Individual causes of death in Japan are about as prevalent now as in this country about 1900. For example, the United States tuberculosis death rate now is 45 per 100,000. In Japan in 1937 the rate was 204, closely resembling our tuberculosis death rate in 1900 of 196. The picture is similar for diarrhea and enteritis. America has more than twice as many men to draw on in the military age group as Japan—25 million men from 20 to 34, as against Japan's 11 million in this age group. Science News-Letter, January 17, 1942.

One small straw that points a dreadful wind. During the first two years of the war (England) deaths from tuberculosis increased in Glasgow about 41 per cent. The 1941 record shows no improvement. Overwork, strain, ill-spent leisure are thought to be responsible for the rise. S. Laidlaw, M.D. and D. MacFarlane, M.D. British Med. Jour., September, 1941.

See the Borden Exhibit at the Charlotte Meeting

Borden's scientifically designed infant foods at Booth No. 6: BIOLAC, NEW IMPROVED DRYCO (quicker solubility and more vitamins A and D), MULL-SOY (for infants allergic to milk), BETA LACTOSE, KLIM, MERRELL-SOULE POWDERED MILKS and BORDEN'S SILVER COW IRRADIATED EVAPORATED MILK.

AUXILIARY

AUXILIARY PLANS FOR STATE CONVENTION

To Charlotte doctors' wives, in this year of 1942, has come "the good privilege" of entertaining the Auxiliary to the Medical Society of the State of North Carolina.

Thoughts of this privilege have brought new life to the Auxiliary of the Mecklenburg County Medical Society, which, at this date, shows in its records proof of three interesting, well-attended luncheon meetings held this winter.

This Auxiliary, and the wives of all Mecklenburg County doctors, now extend to each and every member of the Auxiliary to the State Medical Society heartiest greetings and a most cordial invitation to attend the Twentieth Annual Session of the Auxiliary to the State Medical Society, May 11, 12, and 13.

We breathe and feel every minute the weight of the war clouds, but, even more *because of*, rather than *in spite of* this, do we press our invitation upon you and hope you will accept.

A former president of the Auxiliary to the American Medical Association said that we must "keep reasons and objectives for our organization constantly before us. One of our greatest services is to participate in Public Health Work." The chief aim of the Auxiliary for all time, and for this year, particularly, is the betterment of the health standards and of the actual health of men, women, and children of our land—and there is no better way in which we can serve our country than to follow this aim. The successful outcome of the war for the Allies depends greatly on the health of the men we send to the front and the health of the rest of us, who keep the home fires burning. This getting together, in convention, will prove, we believe, an inspiration that will go far toward keeping this aim before us, preparing us for, and carrying us through, trying times that may come. Won't you come to Charlotte and partake of the hospitality so gladly offered? Let us, wives of doctors, with so many like interests, spend together a season of fellowship, cementing old friendships and making new friends. A warm welcome awaits you.

Tentative plans for the Convention days, May 11-13, follow:

Members of the Hospitality Committee will be in the lobby of the Charlotte Hotel to greet, and give information to in-coming guests.

Rules and Procedure

1. Register on arrival. Present 1941-1942 membership card or pay $1.00 registration fee, and receive badge.
2. Wear badge to all functions.
3. Please register promptly for various events.
4. Visiting ladies are asked to use their own cars if convenient.

Auxiliary General Chairman of Convention

Mrs. Henry L. Sloan

Convention Committees

Hospitality: Mrs. Joseph Elliot, Chairman; Mrs. Aubrey Hawes, Mrs. O. D. Baxter, Mrs. Robert H. Lafferty, Mrs. Brodie Nalle, Mrs. Luther Kelly, Mrs. Andrew Blair, Mrs. R. B. McKnight, Mrs. Wm. Allan, Mrs. W. S. Rankin, Mrs. Vann Matthews.

Bingo Party: Mrs. Graham Reid, Chairman; Mrs. James Alexander, Mrs. L. R. Teasdale, Mrs. Robert T. Ferguson, Mrs. L. C. Todd, Mrs. Jasper S. Hunt, Mrs. W. B. Bradford, Mrs. W. Z. Bradford, Mrs. W. T. Gilmore.

Luncheon: Mrs. Raymond Thompson, Chairman; Mrs. Alonzo Myers, Mrs. Addison Brenizer, Mrs. L.D. McPhail, Mrs. Robert McKay, Mrs. C. L. Nance, Mrs. C. C. Massey, Mrs. Heath Nisbet, Mrs. G. H. Petteway.

Tea: Mrs. Ed Wannamaker, Chairman; Mrs. O. L. Miller, Mrs. E. W. Franklin, Mrs. W. E. Daniel, Mrs. Paul W. Sanger, Mrs. W. R. Pitts, Mrs. A. A. Barron, Mrs. Hamilton McKay, Mrs. Elias Faison, Mrs. Preston Nowlin, Mrs. Allan Tuggle.

Transportation: Mrs. Harry Winkler, Chairman; Mrs. V. K. Hart, Mrs. John Stuart Gaul, Mrs. A. B. Choate, Mrs. Frank C. Smith, Mrs. Chas. H. Gay, Mrs. John P. Kennedy, Mrs.

Chas. W. Robinson, Mrs. Claude B. Squires, Mrs. Fred E. Motley, Mrs. D. G. Welton, Mrs. J. Rush Shull, Mrs. Marvin Scruggs, Mrs. Wm. F. Martin, Mrs. J. B. Hamer, Mrs. E. R. Hipp, Mrs. Preston White, Mrs. Hunter Jones, Mrs. L. B. Newell, Mrs. C. N. Peeler, Mrs. J. Lester Ranson, Mrs. R. A. Moore, Mrs. H. L. Newton.

Mint Museum: Mrs. W. B. Mayer, Chairman; Mrs. J. W. Gibbon.

AUXILIARY TO THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

Twentieth Session

OFFICERS

President—Mrs. Sidney Smith.....Raleigh
President-Elect—Mrs. R. A. Moore....Winston-Salem
First Vice-President—Mrs. C. R. Hedrick.....Lenoir
Second Vice-President—Mrs. J. R. Terry....Lexington
Third Vice-President—Mrs. J. S. Hooker..Chapel Hill
Chairman of Past Presidents—Mrs. P. P. McCain
Sanatorium
Corresponding Secretary—Mrs. J. C. Knox....Raleigh
Recording Secretary—Mrs. Harry Winkler
Charlotte
Treasurer—Mrs. E. C. Judd.....Raleigh

COUNCILORS

First District—Mrs. Thomas L. Carter.....Gatesville
Second District—Mrs. K. B. Pace.....Greenville
Third District—Mrs. D. M. Royal.....Salemburg
Fourth District—Mrs. C. F. Strosnider.....Goldsboro
Fifth District—Mrs. W. T. Rainey.....Fayetteville
Sixth District—Mrs. P. G. Fox.....Raleigh
Seventh District—Mrs. G. Aubrey Hawes....Charlotte
Eighth District—Mrs. E. T. Harrison.....High Point
Ninth District—Mrs. James W. Vernon....Morganton
Tenth District—Mrs. D. I. Campbell King,
Hendersonville

STANDING COMMITTEES

Program—Mrs. Joseph A. Elliott.....Charlotte
Public Relations—Mrs. Wingate Johnson
Winston-Salem
Legislative—Mrs. J. Buren Sidbury.....Wilmington
Press and Publicity—Mrs. Verne S. Caviness
Raleigh
Bulletin—Mrs. Ben Kendall.....Shelby
Hygeia—Mrs. W. G. Byerly.....Lenoir
Memorial—Mrs. George W. Mitchell.....Wilson
Historian—Mrs. J. Roy Hege.....Winston-Salem
Exhibits—Mrs. Alfred A. Kent, Jr.....Granite Falls
Research—Mrs. Rigdon Dees.....Greensboro
Scrapbook—Mrs. Ben F. Royal.....Morehead City
Jane Todd Crawford Memorial—Mrs. F. R. Taylor
High Point
National Defense—Mrs. Thomas Leslie Lee..Kinston
Auditor—Mrs. R. S. McGeachy.....New Bern
Nominations—Mrs. George M. Cooper.....Raleigh
Convention—Mrs. Henry L. Sloan.....Charlotte
Advisory Board—Dr. Caroline McNairy.....Lenoir
Councilor to Southern Medical Auxiliary
—Mrs. J. B. Sidbury

PROGRAM

AUXILIARY TO THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

Headquarters: Hotel Charlotte

MONDAY, MAY 11

1-6 p.m. —Registration ....Hotel Charlotte
1:30 p.m.—Luncheon meeting for Executive Committee and Board of Directors, Hotel Charlotte, courtesy Mrs. Sidney Smith, Raleigh, State President.
8:30 p.m.—Bingo Party....Hotel Charlotte

TUESDAY, MAY 12

9 a.m. —Executive Board Meeting, Chinese Room, Hotel Charlotte.
10:30 a.m.—Annual State Meeting—Charlotte Woman's Club.
*1 p.m. —Luncheon—Charlotte Woman's Club (Fee $.75)
Mrs. Roscoe E. Mosiman of Seattle, Washington, President of Woman's Auxiliary to American Medical Association, Guest Speaker.
*2:30 p.m.—Garden Pilgrimage followed by tea at the home of Mrs. E. J. Wannamaker, 1134 Linganore Place.
7 p.m. —Joint banquet with Medical Society.
10 p.m. —Annual Medical Society Ball.

WEDNESDAY, MAY 13

9 a.m. —Post-Convention Executive Board Meeting — Mrs. R. A. Moore, Presiding — Chinese Room.
*10 a.m. —Visit to Mint Museum of Art. Coffee hour at the home of Mrs. Joseph A. Elliott, 2700 Sherwood Avenue.

* Please register for these events.

PROGRAM

ANNUAL STATE MEETING

Tuesday, May 12, 1942, 10:30 A. M.
Charlotte Woman's Club
Mrs. Sidney Smith, President, Presiding

Call to Order, Mrs. Sidney Smith.....Raleigh
Invocation, Mrs. George W. Mitchell.....Wilson
Memorial Service
Reports of Officers:
President, Mrs. Smith
First Vice-President and State Chairman of Organization—Mrs. Clyde R. Hedrick.....Lenoir
Introduction of District Councilors
Second Vice-President and Chairman of McCain-Stevens Beds—Mrs. J. R. Terry.....Lexington

Third Vice-President and Chairman Student Loan Fund—Mrs. John S. Hooker.....Chapel Hill
Treasurer, Mrs. E. C. Judd.....Raleigh
Announcement of Awards
Chairman of Past Presidents, Mrs. P. P. McCain Sanatorium
Chairman Advisory Board, Dr. Caroline McNairy Lenoir
Recommendations of Board of Directors
Recording Secretary—Mrs. Harry Winkler Charlotte
Introduction of Chairmen of Standing Committees
Introduction of Presidents of County Medical Auxiliaries
Reports:
Councilor for Southern Medical Auxiliary
Chairman of Revisions, Mrs. J. Buren Sidbury Wilmington
Delegate to National Auxiliary —Mrs. Harry Winkler, Charlotte
Courtesy Committee
Greetings from the Medical Society of the State of North Carolina—Dr. F. Webb, Griffith, President Asheville
Unfinished Business
New Business
Report of Nominating Committee —Mrs. G. M. Cooper, Chairman, Raleigh
Election and Installation of Officers —Mrs. P. P. McCain, Presiding
Inaugural Remarks—Mrs. R. A. Moore Winston-Salem
Announcements
Adjournment
Luncheon

Visit of National President

Mrs. Roscoe E. Mosiman of Seattle, Washington, President of the Auxiliary to the American Medical Association, has signified her intention of attending the annual State meeting of the North Carolina Auxiliary at the invitation of the State Board of Directors. Since conditions are extremely unsettled on the West Coast it is not possible for Mrs. Mosiman to say definitely that she will be able to travel across the United States for this meeting. However, she has graciously responded to North Carolina's invitation and writes: "Nothing would give me greater pleasure than attending your meeting. I have looked forward to being with you . . . . and I hope to be able to attend your luncheon meeting of May 12 and discuss some of the problems which confront us."

The State Auxiliary will have its annual session on Tuesday morning, May 12, at 10:30 o'clock at the Charlotte Woman's Club. Officers will attempt to make the meeting brief and informative. A luncheon will follow, at which time Mrs. Mosiman will be guest speaker.

Mrs. Holcombe Is Guest of Wake County Auxiliary

The Auxiliary to the Wake County Medical Society was honored to have as its guest speaker in March, Mrs. V. E. Holcombe of Charleston, West Virginia, immediate past president of the National Auxiliary. The meeting was held at the Woman's Club in Zebulon with Mrs. A. C. Campbell, of Raleigh, president of the Wake County Auxiliary, presiding.

During her visit in North Carolina, Mrs. Holcombe was the guest of Mrs. Sidney Smith, State Auxiliary President, at her home in Raleigh. Mrs. Holcombe is this year devoting her time to civilian defense work. She is Chairman of Volunteer Service for the Charleston Chapter of the American Red Cross and is particularly interested in the work of the Nurses' Aide Corps of the Red Cross, having organized a series of successful classes in Charleston hospitals this winter. While in Raleigh Mrs. Holcombe addressed a student group preparing to become Nurses' Aides at Rex Hospital and was presented in a radio broadcast over WPTF, speaking in behalf of this branch of Red Cross volunteer service.

Announcement

A few more months, and the members of the Woman's Auxiliary to the American Medical Association will be arriving in Atlantic City, New Jersey, for their Annual Convention, June 8-12.

Have you made your reservations? If not, send your request *at once* to Haddon Hall, Atlantic City, New Jersey.

Tuberculosis occupies one of the first places in the disorganization of family life, both socially and economically. It breaks up homes, separates families, destroys husband-wife and parent-child relationships; and it renders many of its victims incapable of resuming their places in society. Economically the cost of this disease to the community is tremendous. It costs a community from $2,000 to $5,000 to care for each case of diagnosed tuberculosis, depending upon the stage of advancement. It has its highest incidence during the most productive years of life, and the financial loss to the patient and to the country as a whole amounts to millions of dollars annually. From an epidemiological standpoint, the disease has the greaest incidence of any disease, with the possible exception of gonorrhea and syphilis.—Grace M. Longhurst, R. N., Amer. Jour. of Nursing, Jan. 1942.

BOOK REVIEWS

Clinical Hematology. By Maxwell M. Wintrobe, M.D., Ph.D., Associate in Medicine, Johns Hopkins University; Associate Physician, Johns Hopkins Hospital. 792 pages, with 167 engravings and 7 colored plates. Price, $10.00. Philadelphia: Lea & Febiger, 1942.

Our knowledge of hematology has been so widened in recent years that the subject is one of great complexity. Dr. Wintrobe, one of the assiduous workers in the field, presents in this book a comprehensive and authoritative survey of the subject. The efficacious management of any patient presenting evidence of a hematological disorder requires an accurate diagnosis. The laboratory methods necessary for arriving at such a diagnosis are explicitly described, and detailed clinical accounts of the various hematological disorders and the means available for their management are given. The book is well organized and clearly written. Bibliographic references to 2400 papers and monographs are included.

Diseases of Metabolism. Edited by Garfield G. Duncan, M.D., Chief of Medical Service "B", Pennsylvania Hospital; Associate Professor of Medicine, Jefferson Medical College, Philadelphia, Penn. 985 pages. Fully illustrated including 7 color plates. Price $12.00. Philadelphia: W. B. Saunders Company, 1942.

The material on the different phases of metabolism has been compiled by fifteen different authors. The chapters on certain major metabolic problems such as obesity and diabetes are excellently written from the standpoint of the practicing physician. The discussion on gout consumes forty-one pages, which seems unduly lengthy. Diseases of the thyroid are not discussed in a separate chapter but are covered under introductory considerations and under iodine metabolism, a total of thirty-three pages being devoted to this important subject. Many of the sections such as that on protein metabolism, cover much of the experimental work on the subject, with discussions of the structural formulae. The bibliography is well selected. On the whole the volume well fills its intended function as a text for practitioners.

Diabetes Mellitus. By Zolton T. Wirtschafter, M.D., Clinician in Charge, Clinic for Diabetes, Department of Medicine, Mount Sinai Hospital, Cleveland; Visiting Physician, Department of Medicine, Cleveland City Hospital; Clinical Instructor in Medicine, School of Medicine, Western Reserve University; and Morton Korenberg, M.D., Former Fellow, May Institute of Medical Research, The Jewish Hospital, Cincinnati; Medical Resident, Jewish General Hospital, Montreal. 185 pages. Cloth, $2.50. Baltimore: The Williams and Wilkins Company, 1942.

A large part of the book is a compilation of one sentence summaries of articles in the literature covering all phases of diabetes. There is little original work included. This volume might prove helpful to students but would be of little value to the practicing physician. The bibliography and format are excellent.

The Blood Bank and the Technique and Therapeutics of Transfusions. By Robert A. Kilduffe, Director, Laboratories, Atlantic City Hospital; and Michael DeBakey, Assistant Professor of Surgery, School of Medicine, Tulane University of Louisiana. Price, $7.50. St. Louis: The C. V. Mosby Company, 1942.

The authors have successfully attempted in this book to collect together in a comprehensive manner a vast amount of material concerning blood and plasma transfusions, the technical methods used and the indications and contraindications for the administration of blood serum and plasma. The book begins with a historical chapter, which is followed by a chapter on the indications and contraindications. The greater part of the book has to do with the technique of blood typing, with compatibility tests, the blood and plasma bank, the preparation of dried plasma, and methods and technique of transfusions. Each chapter is supported by a voluminous bibliography which covers all the literature through July, 1941. It is an excellent book and ought to find wide usefulness both in laboratories and hospitals and among the medical profession generally.

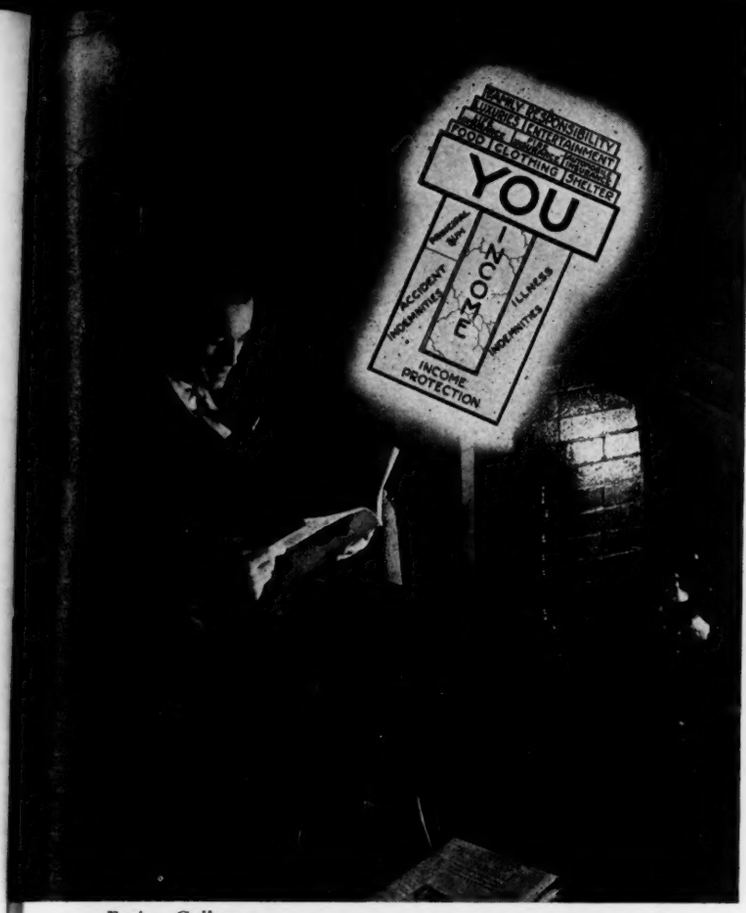
Symposium on Military Medicine, The Medical Clinics of North America, Vol. 25. Illustrated. Cloth, $12.00 per clinic year, paper, $6.00 per clinic year. Philadelphia: W. B. Saunders Company, 1941.

This volume is composed of a number of short individual articles on various phases of disease in relation to the peculiar problems of military medicine. Many of the authors are in the regular armed forces, and the remainder are in the organized reserve. The material should prove especially interesting to those physicians who are unfamiliar with the organization of the medical departments of the armed forces. It is not in any sense a hand-book to be taken into the field.

Infantile Paralysis, 1941. A Symposium. 239 pages, with illustrations. Price, $1.25. New York: The National Foundation for Infantile Paralysis, Inc., 1941.

This book is composed of a series of six lectures given at Vanderbilt University by Drs. Paul F. Clark, Charles Armstrong, Thomas M. Rivers, Ernest W. Goodpasture, John R. Paul, and Frank R. Ober. Each is an accepted authority in his particular field. The lectures deal with the history of poliomyelitis; the etiology; immunological and serological phenomena; pathology and pathogenesis; epidemiology; and treatment. A comprehensive bibliography is included.

The purpose of these lectures is "to bring together a resume of the present day knowledge of this disease" so that those interested may learn what is known about it, what false leads have been followed in investigations concerning it, and what promising studies should be carried further. This purpose has been fulfilled, and the book should be of value to anyone interested in poliomyelitis, whether from a clinical or a research viewpoint.



Ewing Galloway

AS YOU READ GIVE SOME THOUGHT TO **INCOME.**

AN APPLE A DAY—

Is not enough for a doctor's protection!

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It has been our privilege during the past two and one-half years to insure over 800 members of the North Carolina Medical Society under our Special Disability policy, which covers every type of sickness or accident.

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